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PRIMARY CYST (MESOTHELIAL) OF THE DIAPHRAGM

REPORT OF A CASE

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PRIMARY cysts of the diaphragm are rarely reported in the literature. In 1950, Samson and Childress[1] reviewed the literature on primary tumors of the diaphragm. Of 35 tumors recorded, only three were cysts. In 1949, Aufses and Oseasohn[2] reported a mesothelial diaphragmatic cyst and stated that they were able to find only five other recorded cases. In 1951, Cruickshank and Cruickshank[3] reviewed the literature and reported two diaphragmatic mesothelial cysts of their own, bringing the total to eight primary cysts of the diaphragm. We wish to record another.

CASE HISTORY

Z. A., a 57-year-old Negro man, was admitted to the hospital on Oct. 12, 1951, complaining of recurring attacks of left upper quadrant abdominal pain, pain the right shoulder and right infraclavicular regions, and shortness of breath. All symptoms were precipitated by exertion and alleviated by bed rest. At times he complained of a dry, nonproductive cough. There was no hemoptysis. These symptoms had been present for twenty years.

Roentgenograms and fluoroscopy of the chest revealed a soft-tissue tumor mass in the left leaf of the diaphragm which moved synchronously with the diaphragm. An old roentgenogram of the chest, taken elsewhere in 1931 for similar complaints, was available and showed the same mass present, the size having increased moderately in twenty years. At this time the patient refused further diagnostic procedures and left the hospital.

In the next two years he was admitted twice with similar complaints. He was readmitted in September, 1953, complaining of severe upper left quadrant abdominal pain and dyspnea.

Physical examination revealed a tall, thin, asthenic Negro man, 57 years of age, who did not apear to be acutely ill. The blood pressure was 118/84. The heart was normal. The chest moved symmetrically, expansion was equal on both sides, and the lungs were

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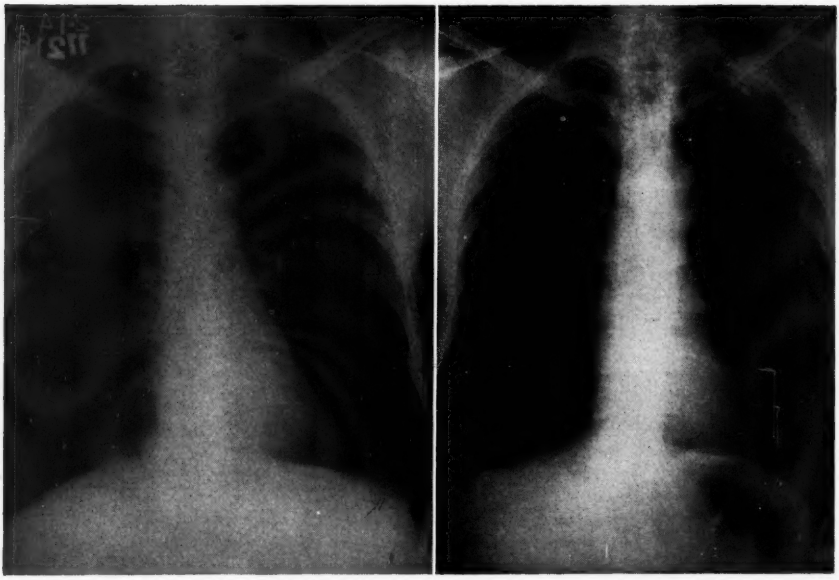

Fig. 1. Fig. 2.

Fig. 1.—Film taken in 1931 showing soft-tissue tumor mass in left leaf of diaphragm.
Fig. 2.—Film taken in August, 1953, showing moderate increase in size of the tumor.

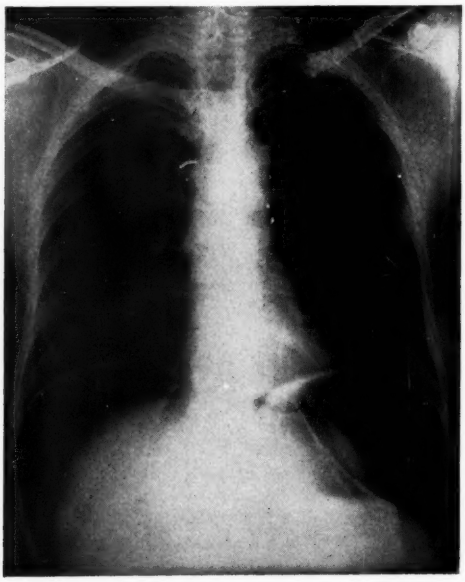

Fig. 3.—Diagnostic pneumoperitoneum (September, 1953) showing the tumor to be intra-diaphragmatic.

normal to percussion and auscultation. There was slight tenderness on deep palpation in the upper left quadrant of the abdomen with increase in pain on compression of the lower rib cage. No other abnormalities were noted.

Fig. 4.

Fig. 5.

Fig. 4.—Appearance of diaphragmatic cyst at surgery before excision.
Fig. 5.—Film taken at surgery showing defect in diaphragm after excision of cyst. Inferiorly the diaphragmatic peritoneum is intact.

Fig. 6.—Appearance of cyst after excision.

On bed rest all symptoms quickly subsided. There were no abnormal laboratory findings. Electrocardiogram, gastrointestinal series, and barium enema were normal. A bronchoscopic examination was negative for pathology. A diagnostic pneumoperitoneum

revealed a well-rounded, smooth, soft-tissue mass 6 by 5 by 4 cm., located in the left leaf of the diaphragm, moving synchronously with the diaphragm. Preoperatively a diagnosis of benign tumor (cyst vs. lipoma) of the diaphragm was made. (Figs. 1, 2, and 3.)

On Oct. 13, 1953, a thoracotomy was done through the bed of the left eighth rib; and a large, paper-thin, walled cyst measuring 6 by 5 by 4 cm. was found anteriorly and laterally in the dome of the diaphragm. There were no adhesions present. The cyst was easily removed by sharp dissection as the diaphragmatic musculature was completely absent in this area. On removal of the cyst a defect was left in the diaphragm, being covered inferiorly by diaphragmatic peritoneum only. This defect was closed with interrupted mattress sutures of 00 black silk. The chest was closed without drainage. The patient had an uneventful recovery and has been asymptomatic to date.

The cyst was filled with clear, straw-colored fluid. The wall was lined by a single layer of flat mesothelium and strands of fibrous tissue. (Figs. 4, 5, 6, and 7.)

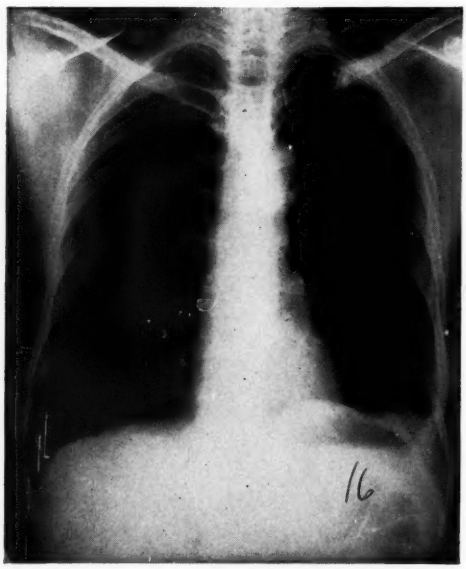

Fig. 7.—Postoperative film of chest (February, 1954.)

DISCUSSION

Primary tumors of the diaphragm are rare. Up to the present time, there were only eight other primary cysts of the diaphragm recorded. Many other mesothelial cysts, i.e., "celomic cysts," have been recorded. We believe with Cruickshank and Cruickshank[3] that these cysts are comparable and form one of a series of cysts arising in different anatomical positions from various parts of the mesothelial lining of the celomic cavities. Celomic cysts are to be differentiated from cysts arising by different embryologic processes from different embryologic structures, i.e., bronchogenic, mesenteric, lymphatic, etc.

There are no true diagnostic signs and symptoms. Many are asymptomatic. The symptoms are varied but tend to follow a pattern of diaphragmatic irritation. Some symptoms, as referred pain to the shoulder, neck, or back, are due

to involvement of branches of the phrenic nerve. These tumors may cause cough, dyspnea, and hemoptysis by compression of the lower lobe of the lung, and effusion by irritation. Gastrointestinal symptoms have been encountered. Depending upon their location, cardiac symptoms may be present. Samson and Childress[1] report clubbing of the fingers, and cardiac symptoms due to the pressure of what amounted to a pulmonary arteriovenous fistula in a malignant tumor of the diaphragm. In diagnosis, a loculated empyema may be troublesome.

SUMMARY

1. Primary tumors of the diaphragm are rare. Benign tumors are slightly more prevalent than malignant tumors.

2. Diaphragmatic cysts may be asymptomatic. When symptoms are present, they are due to size and position of the tumor. The usual symptoms are cough, dyspnea, and occasional hemoptysis, with referred pain to the shoulders and back.

3. Diagnosis is made by roentgenograms and fluoroscopy, utilizing pneumoperitoneum and pneumothorax when necessary.

4. Treatment is excision.

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RADICAL EXCISION OF MALIGNANT CHEST WALL TUMORS

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PRIMARY or secondary malignant neoplasms occasionally involve the deep structures of the chest wall. Sarcomas of various types account for most of the primary lesions. Recurrences following radical mastectomy for carcinoma of the breast comprise the majority of secondary tumors, and direct extension from the lung is not infrequent. Treatment of these lesions until recently has been unsatisfactory, and even today many surgeons are reluctant to undertake radical excision, and not infrequently rely upon palliative measures. Any attempt at surgical cure should consist of wide radical extirpation.

When resection of the bony chest wall is accomplished and the defect simply covered by soft tissue, instability is certain to occur. Extensive paradoxical motion, especially in the presence of a mobile mediastinum, can lead to serious complications in the immediate postoperative period. For this reason, many surgeons in the past have avoided surgery in this location or compromised the extent of excision required.

Numerous attempts to stabilize large defects, using autogenous grafts of bone, cartilage, and fascia have been described. These methods have been quite successful in some hands, but, as a general rule, are time consuming. Essentially, a second operation is performed on a patient who has already undergone extensive surgery.

In 1947, one of us (J. M. B.)[1] attempted prosthetic repair of large chest wall defects using tantalum plate which was molded and sutured to the surrounding bony structures with wire. Good stability was obtained in the immediate postoperative period, but the plates were poorly tolerated and had to be removed within a few weeks. Since permanent stability is more desirable in these cases, plates were abandoned in favor of tantalum mesh.

We[2] recently published a report on the use of mesh prostheses in two cases. The material was kindly tolerated by the tissues and seemed to give satisfactory long-term support. Other writers appear to share these opinions. Tearing of implanted mesh has been observed by some authors. The acute stresses of constant chest motion have a wearing effect on mesh fibers, and fracture may occur in time. Although tearing has not been detected in any of our cases, the implantation technique has been modified because of these reports. Originally, mesh was sutured to bone and chest wall fascia about the edges of the defect. Two layers of mesh are now employed, and since fixation of mesh to moving bone may predispose to tearing, the material is allowed to overlap the edges of

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the defect about 1 inch and is tacked snugly to fascia with interrupted cotton sutures. It seems reasonable to assume that fascia exerts some cushioning effect when transmitting the stresses of chest wall motion. Stability obtained with this technique has been excellent. Stainless steel mesh with which we have had no experience is claimed to have even less tendency to fracture than tantalum and certainly seems deserving of further trial.

Once the mesh has been secured over the defect, it must be covered by soft tissue. Simple undermining of wound edges, sliding flaps, and pedicle flaps have all been used in different situations. When dealing with anterior chest wall defects, mobilization of the entire opposite breast and underlying pectoral muscle provides a large and convenient donor flap. This procedure is technically quite simple and has provided very satisfactory coverage each time it has been used. There has been no difficulty with wound healing in any of our extensive resections. All, except one in which there was a partial breakdown of a sliding flap, have healed per primam, and in this instance, despite the fact that underlying mesh was exposed, granulation filled the area rapidly and the wound healed uneventfully.

The purpose of the present paper is to present ten consecutive cases of chest wall malignancy treated by radical excision and prosthetic repair. Once the diagnosis of chest wall sarcoma or carcinoma has been established, curability of the lesion should be considered. A careful clinical and roentgenographic search for metastases is in order if one is dealing with a primary lesion, and detectable spread elsewhere must be ruled out in secondary tumors.

The procedure should be limited to cases considered curable, and has not been recommended to any patient as a palliative measure. In those cases where cure was not achieved, however, worth-while palliation was obtained for considerable periods of time.

CASE REPORTS

Primary Carcinoma of Lungs.—

CASE 1.—W. M., a 72-year-old white man, was admitted to the Rhode Island Hospital on April 29, 1948, because of symptoms and x-ray findings consistent with a malignant neoplasm of the lower lobe of the left lung. There was, in addition, x-ray evidence of tumor extension to the adjacent seventh rib.

On May 6, 1948, radical en bloc excision included left lower lobectomy, large segments of the fifth, sixth, seventh, and eighth ribs with the overlying chest wall soft tissues. The chest wall defect was closed by suturing a tantalum plate to the surrounding ribs with wire. Pathology report was a large bronchiogenic carcinoma measuring 7 by 4.5 by 5 cm. with invasion of the pleura and the adjacent sixth and seventh ribs.

Postoperatively, chest wall stability was very satisfactory, but there was considerable drainage from the wound. Six weeks later, because of persistent drainage and local pain, the plate was removed.

The patient experienced complete relief of symptoms and rapidly gained weight: When seen on the last follow-up visit on April 9, 1954, six years after resection (Fig. 1), he stated he led an active life and felt fine. There were no clinical or x-ray signs of recurrence.

CASE 2.—L. M., a 52-year-old white man, entered the Rhode Island Hospital in July, 1950, because of symptoms and x-ray findings consistent with a malignant neoplasm of the upper lobe of the right lung.

In August, 1950, exploration revealed a huge tumor which was firmly attached to the third and fourth ribs. A right pneumonectomy with en bloc excision of the second, third, fourth and fifth ribs and overlying chest wall soft tissues was accomplished (Fig. 2). The defect was closed by suturing tantalum mesh to the chest wall fascia with interrupted cotton. Pathology report was adenocarcinoma of the lung with peribronchial lymph node metastases.

Recovery was uneventful. (Fig. 3.) He experienced dramatic relief of symptoms and was able to resume his work in a textile mill for four months. Weakness and weight loss then recurred and he was referred to a Veterans Hospital for further study. He died one year after surgery of recurrent disease. The chest wall was described as very stable by physicians in attendance prior to his death.

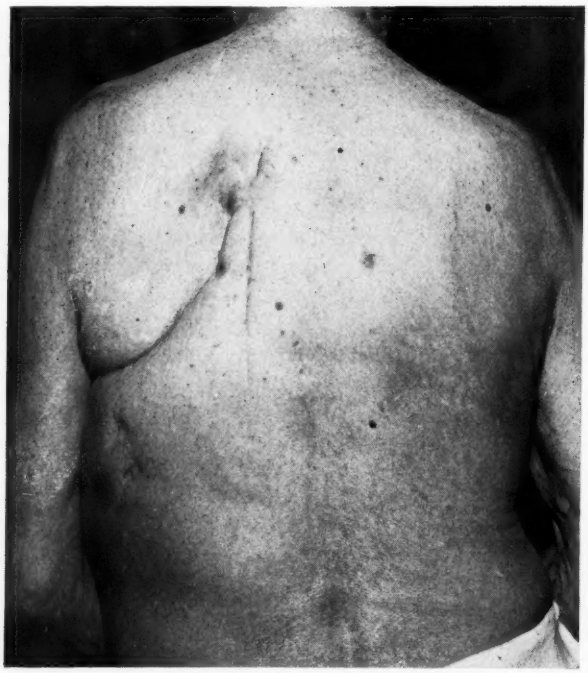

Fig. 1.—Case 1. Photograph of patient six years after operation.

Recurrent Tumors of the Breast.—

CASE 3.—L. S., a 54-year-old white housewife, entered the Rhode Island Hospital on Dec. 4, 1949. Seven years before, a left radical mastectomy had been performed for carcinoma of the breast with axillary metastases. She did well until sixteen months prior to admission when a mass appeared on the left anterior chest wall. X-ray treatments were recommended by her physician. There was temporary tumor regression, but three months before admission the mass again began to grow. It became extremely painful, and she lost 45 pounds. Hormones and more x-ray therapy were prescribed without benefit: She was then referred to the hospital. (Fig. 4.)

Physical examination revealed a 4 by 8 cm. firm mass which was fixed to the sternum and left clavicle. On Dec. 6, 1949, en bloc excision was done which included the upper third of the sternum, wide segments of the left clavicle and upper three ribs, the underlying phrenic nerve, and all overlying soft tissues. The chest wall defect was repaired by suturing a tan-

Fig. 2.

Fig. 3.

Fig. 2.—Case 2. Specimen of right lung with adjacent wall removed at operation.

Fig. 3.—Case 2. Appearance of wound nine months after surgery. (Reproduced by permission of The New England Journal of Medicine.)

Fig. 4.—Case 3. Appearance of lesion before operation.

Fig. 5.—Case 3. Photograph of patient 4 years and 5 months after surgery, illustrating the use of the opposite breast and pectoral muscle as a pedicle flap. Note that nipple has been excised.

talum plate to the surrounding bony structures with wire. The right breast and pectoral muscle were then mobilized and swung as a flap graft to supply soft tissue coverage. Pathology report was adenocarcinoma of the chest wall invading the sternum and left sternoclavicular joint.

Her immediate postoperative course was uneventful. One month after surgery, however, she was readmitted to the hospital because of severe local pain and recurrent pleural effusions requiring thoracenteses. The plate was removed. There was no further effusion or pain, and the wound quickly healed.

She rapidly regained her lost weight and resumed her household duties. When seen on last follow-up visit on April 8, 1954, over four years later, she was leading an active, unrestricted life and had no complaints. There were no clinical or x-ray signs of recurrent tumor. (Fig. 5.)

CASE 4.—H. H., a 72-year-old white widow, entered the Rhode Island Hospital in June, 1950. Ten years before, a left radical mastectomy was done for carcinoma of the breast. Two years prior to admission, a lump appeared on the left anterior chest wall. Tablets were prescribed by her physician, but the mass slowly enlarged. Three months before entry, the mass began to grow rapidly and finally became so painful that hospitalization was recommended.

Physical examination revealed a 4 cm. firm tumor which was fixed to the left second costal cartilage.

En bloc excision included the upper two-thirds of the sternum, wide segments of the first three ribs bilaterally, the medial third of the left clavicle, and all overlying tissues. The mass extended deeply into the mediastinum. Segments of the upper lobe of the left lung and left phrenic nerve were removed to encompass the growth. The chest wall defect was repaired by suturing a tantalum plate to the surrounding bony structures with wire. A circumferential incision was then made about the lower two-thirds of the right breast and pectoral muscle, which were swung across the chest to supply soft tissue coverage.

Pathology report was recurrent adenocarcinoma of the chest wall. Her wounds healed well and the immediate postoperative course was benign. Three weeks after surgery, however, the wound suddenly opened and bled profusely. She was readmitted to the hospital, and the plate was immediately removed.

During the next year, the patient resumed her usual activities and had no complaints. In July, 1951, chest pain recurred and a nodule was felt at the upper margin of the sternal stump. At re-exploration, numerous nodular recurrences were found in the adjacent ribs, sternum, and underlying mediastinum. All visible tumor was excised, but it was felt that further radical surgery was hopeless. The soft tissues were closed and no attempt was made to reinforce the chest wall. The pathology report was recurrent adenocarcinoma. Recovery was uneventful, and x-ray therapy was recommended on discharge.

Surprisingly enough, she had complete relief of chest pain following surgery. She was able again to resume her usual activities and has traveled South during the past two winters.

At her last follow-up check in April, 1954, she complained of pain in both hips. X-ray revealed metastases to the pelvic bones. Some relief has been obtained from testosterone, and, in spite of metastases, she at present leads a moderately active life nearly four years after her original operation.

CASE 5.—M. S., a 62-year-old white housewife, entered the Rhode Island Hospital on March 8, 1951, because of a tumor of the right anterior chest wall. Sixteen months previously, a right radical mastectomy had been done for carcinoma of the breast. Nine months later, a lump appeared on the anterior chest wall. This was locally excised and proved to be adenocarcinoma. A week before the present admission, the patient noted another lump in the same location.

Physical examination revealed a firm, fixed, 4 cm. mass beneath the medial portion of the right clavicle.

En bloc excision included the medial half of the right clavicle, wide segments of the first three ribs on the right, the upper third of the sternum, the first three costal cartilages on the left, and all overlying soft tissues. The chest wall defect was repaired by suturing tantalum mesh to the chest wall fascia and cervical fascia above with interrupted cotton sutures. The left breast was then mobilized as in the previous cases for soft tissue coverage (Fig. 6). The pathology report was recurrent adenocarcinoma of the chest wall.

The patient tolerated the surgery well. She received 500 c.c. of blood in the operating room and returned to the ward in good condition. About seven hours later, she went into sudden vascular collapse and died within a few moments.

Autopsy revealed bilateral atelectasis due to aspiration of tracheobronchial secretions. No gross or microscopic evidence of tumor could be found anywhere in the body.

These patients not uncommonly have difficulty coughing up bronchial secretions during the first day or two following surgery. This tragic and preventable death seems to emphasize the importance of the constant and meticulous care one must devote to bronchial toiletry during this period.

Fig. 6.—Case 5. Demonstrating defect and method of mobilizing the opposite breast to repair the soft tissue defect.

CASE 6.—M. M., a 61-year-old white woman, entered the Rhode Island Hospital on April 26, 1951, because of an anterior chest wall mass. The lump had been present for sixteen months and had recently become painful. A right radical mastectomy had been performed seven years previous for carcinoma of the breast.

Physical examination revealed a 5 cm., firm, immovable mass to the right of the sternum in the third interspace.

En bloc excision included large segments of the third, fourth, and fifth ribs bilaterally, the interposed sternum, and all overlying soft tissues (Figs. 7-9). The chest wall defect

was repaired by suturing tantalum mesh to the surrounding fascia with wire. A few wire sutures were encircled about adjacent ribs. Soft tissue closure was accomplished by rotation of the opposite breast as in the previous cases. Pathology report was recurrent adenocarcinoma of the chest wall. The patient's postoperative course was uneventful, and she was discharged ten days after surgery. (Fig. 10.)

During the next year, she carried on with her usual activities and felt very well. In February, 1953, however, she began complaining of weakness and weight loss. She died four months later of widespread metastatic carcinoma.

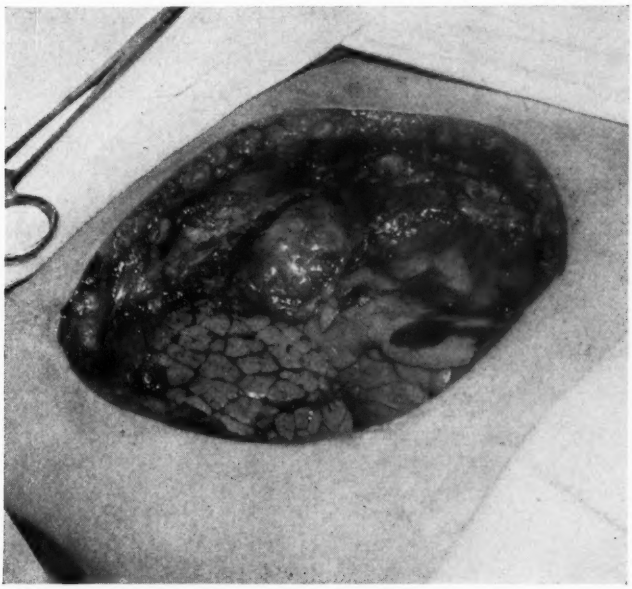

Fig. 7.—Case 6. Defect following excision of lesion. (Photographic Dept., Rhode Island Hospital.)

CASE 7.—N. O., a 69-year-old white housewife, entered the Rhode Island Hospital on May 23, 1951, because of a lump of the right anterior chest wall of two weeks' duration. A right radical mastectomy had been performed two years earlier for carcinoma of the breast with axillary node metastases.

Physical examination revealed a firm, fixed, 4 cm. mass in the right sternoclavicular area.

Radical excision included the medial third of both clavicles, wide segments of the first and second ribs bilaterally, the upper 8 cm. of sternum, and all overlying soft tissues. The chest wall defect was closed with a double layer of tantalum. The mesh was allowed to extend 1 inch beyond the edges of the defect and was sutured to fascia with interrupted cotton. Soft tissue closure was accomplished by rotation of the opposite breast. Pathology report was recurrent adenocarcinoma of the chest wall.

Her postoperative course was uneventful and chest wall stability excellent. Unfortunately, she died at home of a myocardial infarct one month after discharge. Permission for autopsy was not obtained.

CASE 8.—D. T., a 56-year-old white housewife, entered the Rhode Island Hospital on June 21, 1953. She had noticed a lump of the right anterior chest wall for over two years. It had been increasing in size slowly, and recently it had become painful. A left radical mastectomy had been performed in 1935 for carcinoma of the breast.

Fig. 8.

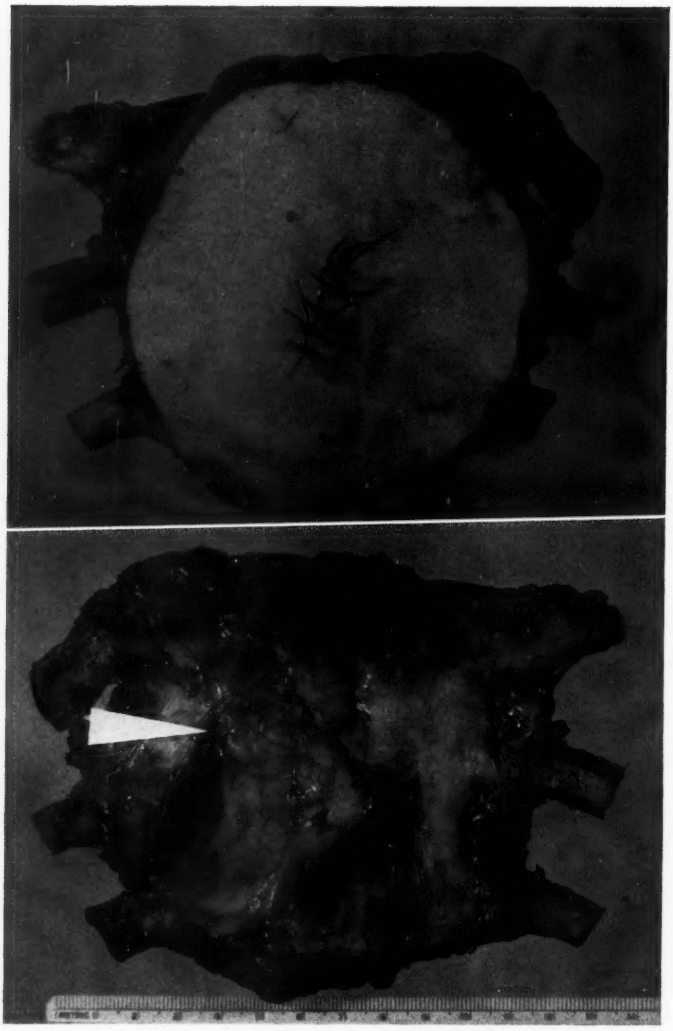

Fig. 9.

Fig. 8.—Case 6. Anterior view of excised specimen.
Fig. 9.—Case 6. Posterior view of specimen. Note tumor bulging on mediastinal surface.

Physical examination revealed a large, firm, fixed mass between the second and third ribs of the right anterior chest wall.

At operation, a 12 by 6 by 4.5 cm. ovoid mass was found to bulge into the pleural cavity. It involved the third rib but was not attached to the underlying structures. Radical excision included the second, third, and fourth ribs from the sternum to their paravertebral attachments, and all overlying soft tissues. The chest wall defect was repaired by suturing a double layer of tantalum mesh to chest wall fascia with interrupted sutures. Mesh was allowed to overlap the edges of the defect for about one inch. (Fig. 11.) Soft tissue closure was accomplished easily by undermining. Pathology report was recurrent adenocarcinoma of the chest wall.

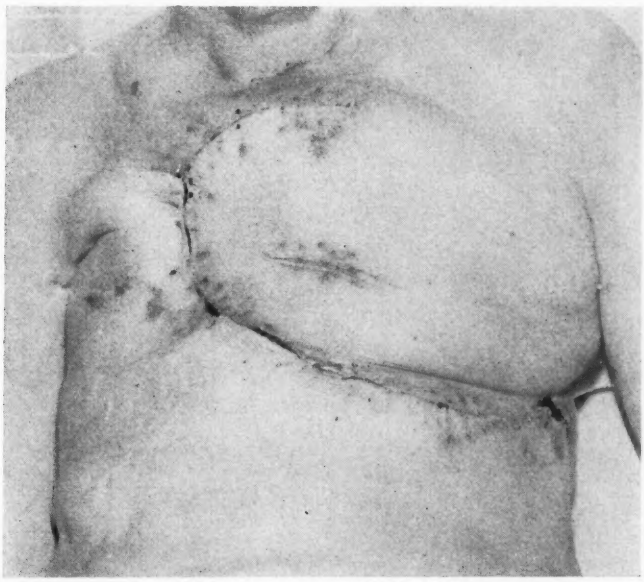

Fig. 10.—Case 6. Appearance of anterior chest wall ten days after operation. (Photographic Dept., Rhode Island Hospital.)

The patient's postoperative course was uneventful. Chest wall stability was excellent. She was discharged on the tenth postoperative day. When seen last on April 2, 1954, she was feeling well, had a good appetite, and was leading an active life. There were no clinical or x-ray signs of recurrence.

CASE 9.—C. T., a 63-year-old white widow, entered the Rhode Island Hospital in February, 1947, because of a rapidly enlarging mass of the anterior chest wall first noted six months before. X-rays revealed extensive generalized Paget's disease. In the manubrium, a 5 to 6 cm., expanding, destructive lesion had eroded the sternoclavicular joints. The mass was visually obvious and very firm on palpation. (Fig. 12.)

Radical en bloc excision included wide segments of both clavicles, the upper three ribs bilaterally, 10 cm. of upper sternum, and all overlying soft tissues.

The chest wall defect was repaired by suturing a tantalum plate to the adjacent ribs with wire. Soft tissue coverage was obtained by rotation of the left breast. Pathology report was osteogenic sarcoma of the manubrium with invasion of the sternoclavicular joints.

Fig. 11.—Case 8. Showing double layer of tantalum mesh in place.

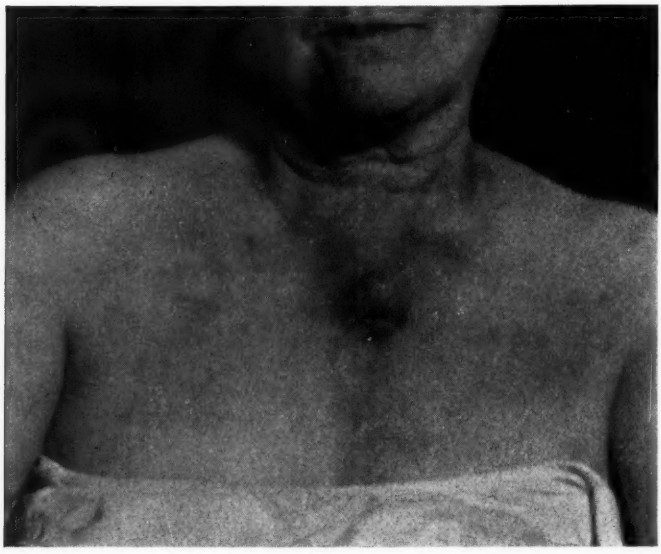

Fig 12.—Case 9. Osteogenic sarcoma of sternum. (From Beardsley; J. Thoracic Surg., March, 1950.) Patient was living without recurrence 7 years and 2 months after radical excision.

The patient's postoperative course was complicated by considerable local pain and drainage from the wound. Three months after surgery, she was readmitted to the hospital because of persistent pain and drainage. The plate was removed. She quickly regained her sense of well-being and the wound healed uneventfully.

When last seen in April, 1954, over seven years post resection, she was leading an active life and had no complaints. There were no clinical or x-ray signs of recurrence.

Fig. 13.—Case 10. Rhabdomyosarcoma of right posterior chest wall.

CASE 10.—M. G., a 77-year-old white widow, entered the Rhode Island Hospital in August, 1950. She complained of an extremely painful grapefruit-sized mass of the right posterior chest wall (Fig. 13). The tumor had been present for six months, was increasing in size rapidly, and she had lost 30 pounds.

Radical excision included large segments of the seventh, eighth, ninth, tenth, and eleventh ribs, some underlying pleura, and all overlying soft tissues (Fig. 14). The chest wall defect was repaired by suturing tantalum mesh to chest wall fascia with interrupted cotton sutures. A few sutures encircled the sixth rib above and the twelfth rib below. Soft tissue coverage was accomplished by utilizing a sliding flap.

Pathology report was rhabdomyosarcoma of chest wall.

The patient was a hypertensive, elderly, debilitated woman. Her convalescence was prolonged and complicated by a partial breakdown of the sliding graft. Despite the fact that underlying mesh was exposed, the area granulated in and healed solidly. She was discharged on the sixty-first hospital day. (Fig. 15.)

During the next two years, she did well. Her weight was maintained, and she was able to perform her usual chores about the house. In August, 1952, however, she began complain-

Fig. 14.—Case 10. Specimen showing amount of chest wall resected.

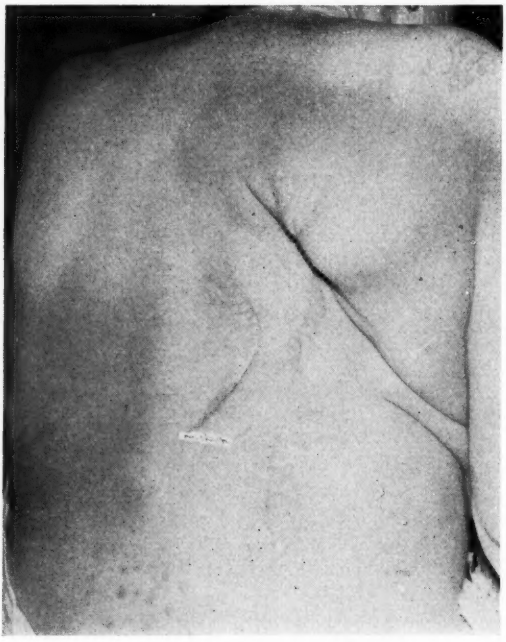

Fig. 15.—Case 10. Patient nine months after surgery. (Reproduced by permission of The New England Journal of Medicine.)

ing of weakness and back pain. Shortly after this, she was sent to a nursing home for care. While there, a large recurrent tumor mass appeared in the presacral area. She ran a progressively downhill course and died in March, 1953. Permission for autopsy was not obtained.

DISCUSSION

Primary carcinoma of the lung which invades adjacent chest wall structures frequently arises in peripheral lung tissues. Hilar encroachment by these growths is less likely than with centrally located lesions, and hence pneumonectomy or lobectomy is generally not technically difficult. Invasion in our two cases was confined to bone and periosteum. Resection of chest wall was limited to rib and intercostal muscles. The overlying soft tissues were not disturbed, and local recurrence did not occur in either case. The chest wall resection adds little time to the procedure and none to the convalescence. In the absence of detectable metastases elsewhere, a reasonable salvage rate should be expected.

Fixation of breast cancer to chest wall has long been considered a sign of inoperability and because of the prevalently taught dogma, many of the above cases were sent to the hospital for terminal disposition. The growths had been observed by the patients themselves an average of fourteen months before referral. Usually a wide circular incision is made about these tumors including a liberal margin of normal skin. As the incision is extended down through all layers of chest wall, carefully isolation and ligation of the intercostal and internal mammary vessels will prevent troublesome hemorrhage. Of six recurrent breast cancers so treated, one is alive and apparently free of disease more than four years after resection. Another has no evidence of recurrence ten months after surgery and, in a third case, there was no gross or microscopic evidence of tumor at autopsy. A fourth patient unfortunately suffered a fatal myocardial infarct shortly after leaving the hospital. Definite recurrence has been observed in only two cases, and one of these obtained palliation of severe chest pain for over three years. Perhaps earlier referral will increase the salvage rate in these cases. Primary malignant tumors occurring on the chest wall are treated by similar wide circular excision. Since these growths are physically obvious and detected at an early stage by the patient himself, cures should be expected in a favorable percentage.

A series of ten cases is, of course, much too small to be of any clinical significance. These cases are infrequently encountered, however, and follow-up reports on large series of surgically treated chest wall malignancies are rarely seen.

Surgery for these tumors has been criticized as mutilating, dangerous, and hopeless. Actually, with prosthetic repair, physical deformity is not marked, and there should be no functional incapacity. One preventable death in ten cases can hardly be used as an argument against the safety of the procedure. All wounds are closed tightly without drainage, and healing per primam has been the rule. One elderly, debilitated woman had a partial breakdown of a graft and a protracted hospital course. Most other patients left the hospital by the tenth postoperative day.

One patient is alive seven years after resection of a large osteogenic sarcoma of the sternum, another six years after resection of an advanced epidermoid

carcinoma of the lung invading the chest wall, and a third four years after excision of a recurrent breast carcinoma that had infiltrated the bony cage.

X-ray therapy rarely if ever leads to permanent cure of malignancies of the nature described above.

Our experience with ten cases with seem to indicate that any chest wall malignancy, which after careful study appears to be self-limited, should be given the benefit of the doubt and widely excised.

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TRAUMATIC HEMOMEDIASTINUM

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DESPITE the current emphasis on lesions of the mediastinum and the increased incidence of injuries involving the thorax, the occurrence of mediastinal hematoma appears to have evoked little interest. It is believed that this complication is more frequent than the paucity of available reports would indicate. For this reason it is felt desirable to review present knowledge regarding hemomediastinum, with particular attention to that following trauma, and to describe an illustrative case.

Significant mediastinal hemorrhage, presumably nontraumatic, has been observed in association with numerous diseases including rupture of syphilitic[1] and arteriosclerotic[2, 3] aortic aneurysms, hemorrhage into parathyroid adenomas,[4] uremia,[5] typhoid fever,[6] and "venular heredofamilial hemorrhagic diathesis."[7] The case of hemomediastinum following "rupture of an atheromatous aorta," reported by Duperrat and Ullmann,[8] would appear on examination of the pathologic data presented by these authors to have been due rather to dissecting aneurysm.

Reported instances of mediastinal hematoma secondary to trauma are almost equally sparse. Körner,[9] in 1916, described the case of a 30-year-old soldier who was injured by an exploding grenade and who developed a large posterior mediastinal hematoma which displaced both trachea and esophagus. Other authors[10] have drawn attention to the association of hematomas of the posterior mediastinum with fractures of the thoracic vertebrae. Mediastinal hematoma following penetrating trauma has been reported by Horn,[11] whose patient, a 21-year-old man, sustained a stab wound of the chest with laceration of the left internal mammary artery. The patient recovered, and serial chest x-rays showed progressive absorption of the hematoma, resolution being complete within forty-one days. Zimmerman[12] has described the occurrence of acute hemomediastinum following a severe lateral compression injury of the chest. Respiration was never seriously embarrassed, conservative treatment was successful, and repeated chest x-rays over a one-month period showed gradual absorption of the hematoma. In the case reported by Schmitt,[13] multiple chest injuries occurred as a result of a motoring accident, a huge mediastinal hematoma being associated with pulmonary parenchymal hemorrhage, hemothorax, and rib fracture. Death occurred unexpectedly fourteen days after injury. Duyster's[14] patient with mediastinal hematoma likewise presented multiple concomitant thoracic injuries. Vincent and associates[15] have reported an instance of fatal hemomediastinum in a 12-year-old child

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who fell 50 meters into a lake. Chest x-ray showed enormous enlargement of the mediastinum. Death supervened forty-eight hours after injury, and autopsy revealed a huge mediastinal hematoma, but the precise site of its origin could not be determined. Further attention has recently been directed to traumatic mediastinal hematoma by Endress,[16] who has described two cases following chest injuries sustained in automobile accidents.

Most standard treatises on roentgen diagnosis accord but brief mention to traumatic mediastinal hematoma. Shanks and Kerley[17] state that it may result from injury to a large vessel in the root of the neck, the blood gravitating inferiorly into the mediastinal areolar tissue. On x-ray the mediastinum is widened and may show straight vertical borders. Schwinz and associates[18] mention that its roentgen differentiation from mediastinitis may be difficult. According to Ritvo,[19] there is usually bilateral widening of the supracardiac shadow, and, if the blood remains confined to the mediastinal region, the margins of the shadow are straight, sharply defined, and smooth in outline.

The following case illustrates many of the clinical and roentgenologic characteristics of traumatic mediastinal hematoma.

CASE REPORT

On Dec. 6, 1953, a 20-year-old sailor was admitted to Framingham Union Hospital* at 4 A.M., shortly after an accident in which he fell asleep at the wheel of his car and struck a tree. He had not been rendered unconscious by the injury. When examined, the patient's chief complaint was difficulty in breathing. Pulse was 120 per minute and blood pressure was 90/70 mm. Hg. There were numerous lacerations, abrasions, and contusions of the face, chest, and lower extremities. Neck veins were slightly distended, and moderate cyanosis was present over the head and neck. A right Horner's syndrome was noted. The trachea was displaced to the left. There were fullness and tenderness over the right upper anterior chest and over the medial aspect of the right clavicle, pressure on which elicited crepitus. The medial end of the right clavicle was movable, and small fragments of bone were palpable in the sternal notch. Subcutaneous emphysema was absent. Auscultation of the lungs was unremarkable except for decreased breath sounds at the right pulmonary base. The right radial pulse was as forceful as the left. Red cell count on admission was 4.56 million, and hemoglobin was 16.6 Gm. per cent. Urinalysis was unremarkable.

Posteroanterior (Fig. 1) and lateral x-rays of the chest showed a dense soft tissue mass involving the superior mediastinum bilaterally, predominantly on the right side. The trachea and presumably the esophagus were displaced to the left. There was a fracture of the second right rib anteriorly. The space between the medial ends of the clavicles was widened, suggesting the possibility of a fracture of the upper sternum. In the lateral projection the superior mediastinum was completely cloudy.

The patient was considered to have a hematoma of the anterosuperior mediastinum, chiefly on the right, secondary to laceration of superior mediastinal veins, possibly the thyroid vein, transverse cervical vein, or smaller tributaries of the right subclavian vein.

All external wounds were cleaned, sutured, and dressed. Parenteral penicillin was administered prophylactically. An infusion of 5 per cent dextrose in water was begun. The patient was less dyspneic when sitting semiupright and was, therefore, maintained in this position. Oxygen was not necessary. Endotracheal aspiration was used to prevent the accumulation of secretions. Four hours after admission blood pressure was 100/70 mm. Hg, and pulse 92 per minute. A small hemoptysis occurred at this time but was not repeated. The clinical condition was improved, and needle aspiration of the hematoma was not considered necessary.

*Service of Dr. Lee G. Kendall.

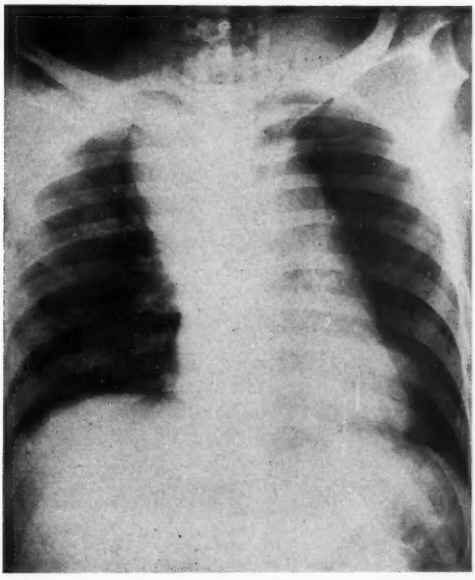

Fig. 1.—Posteroanterior chest x-ray secured upon admission, showing large mediastinal hematoma. The tracheal deviation is evident but bony injuries are not clearly reproduced.

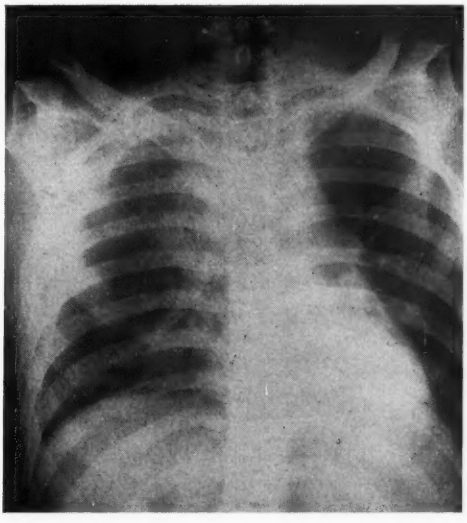

Fig. 2.—X-ray thirty hours after injury. There has been a striking spontaneous decrease in the size of the hematoma. The presence of right pleural fluid is suggested.

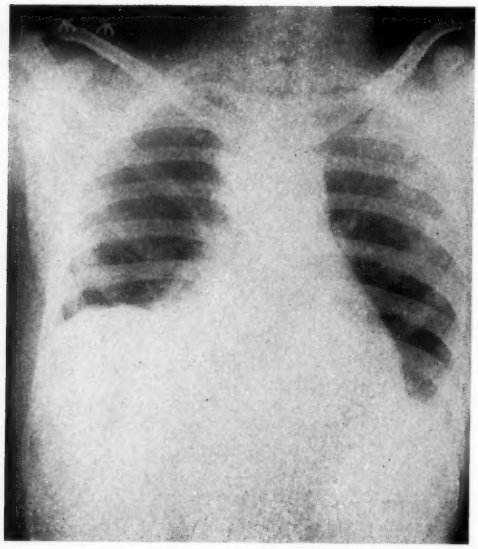

Fig. 3.—Two days after trauma. Further improvement with return of the trachea to the midline and continued regression of the hematoma. Slight elevation of the right hemidiaphragm.

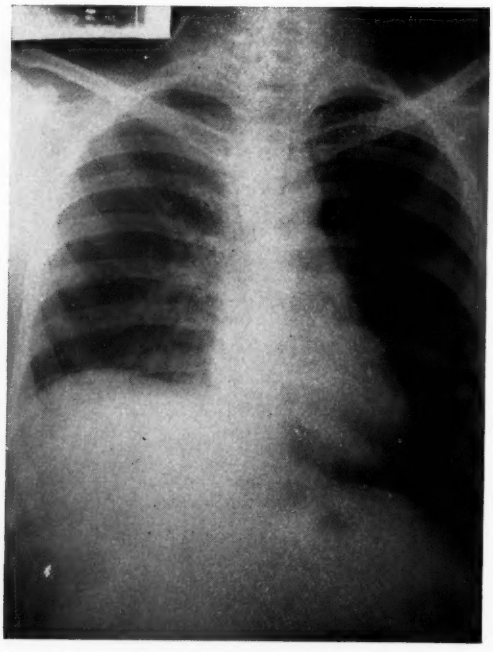

Fig. 4.—Elevation of the right hemidiaphragm is the only significant abnormality seen on this x-ray taken sixteen days after injury. (Courtesy U. S. Naval Hospital, Chelsea, Massachusetts.)

Chest x-ray on Dec. 7, 1953, approximately thirty hours after injury, showed striking regression of the mediastinal hematoma (Fig. 2). There was, however, evidence of thickened pleura and fluid accumulation on the right. On Dec. 8, 1953, chest x-ray showed further narrowing of the mediastinal mass (Fig. 3) and almost complete absorption of the right-sided pleural fluid. The right hemidiaphragm was slightly elevated. The trachea was almost in the midline.

The patient's subsequent course was one of progressive improvement, both clinically and roentgenologically. The Horner's syndrome regressed and dyspnea receded. On Dec. 13, 1953, the patient was transferred to the U. S. Naval Hospital, Chelsea, Massachusetts. Pain in the right sternoclavicular region was the only outstanding complaint at this time. X-rays showed slight separation of the right sternoclavicular joint, and compression and distraction of the joint produced pain. Physiotherapy, with active shoulder-girdle exercise and diathermy, resulted in improvement. Chest x-ray on Dec. 22, 1953, was unremarkable except for elevation of the right hemidiaphragm (Fig. 4). However, inspiration-expiration x-rays, together with fluoroscopy, showed normal diaphragmatic movement bilaterally. On Jan. 14, 1954, the patient was discharged to duty.

(Subsequent investigation of the patient's health record showed that his most recent chest x-ray prior to injury had been secured on March 19, 1952, and indicated no abnormality.)

DISCUSSION

Mediastinal hematoma complicating disease is chiefly of academic interest, both therapy and prognosis being those of the underlying condition. In such instances, unless catastrophic, the development of the hematoma is usually gradual, permitting adequate readjustment of cardiorespiratory dynamics. However, the clinical and roentgenologic findings may require differentiation from other causes of mediastinal enlargement, particularly those amenable to surgical or radiation therapy. In contradistinction, hemomediastinum due to trauma often presents the dual problem of acute blood loss and sudden alteration of cardiopulmonary physiology. As such, it may constitute an immediate threat to the life of the patient.

In view of the present high incidence of crushing chest injuries, especially of the so-called "steering-wheel" type, the apparently rare occurrence of traumatic hemomediastinum is surprising. It is felt, however, that the lesion is actually more frequent than a survey of available reports would suggest. In many instances, for example, it may be considered a relatively unimportant component of a large complex of multivisceral injury. An additional reason is mentioned by Endress,[16] who feels that since x-rays of patients with chest trauma are usually done at short distances and with Bucky technique, the mediastinum is scanned less carefully because of the expected distortion.

The source of the hemomediastinum in instances of blunt injury would appear almost invariably to be venous. In the present case, it was believed to result from laceration of smaller superior mediastinal veins such as the thyroid vein, transverse cervical vein, or smaller subclavian tributaries. With fracture of ribs or sternum, the possibility of injury to intercostal or internal mammary veins must be considered. Fracture of the sternum may also produce copious bleeding from the vascular marrow.[16] In penetrating injuries arterial hemorrhage may be predominant, as in Horn's[11] case of stab wound of the internal mammary artery.

Findings on physical examination in patients with traumatic mediastinal hematoma depend upon its extent, location, and rapidity of accumulation. There is usually obvious outward evidence of injury to the bony thorax. Obstruction to venous drainage may be indicated by distended neck veins. The patient may exhibit marked dyspnea, often favoring the semiupright position for relief. Shock may result from reduction of circulating blood volume, especially should the hematoma rupture into the free pleural space. A transient unilateral Horner's syndrome was noted in the present case. In short, clinical findings may be suggestive but are rarely unequivocal.

The roentgen characteristics of traumatic hemomediastinum have been briefly discussed, most authors emphasizing enlargement of the mediastinal shadow with straight, smooth, sharply defined margins. Endress[16] distinguishes two possibilities. In one, the findings may be most marked on posteroanterior projection with a lateral bulging of the mediastinum being evident. A second type is most apparent in the lateral x-ray, which may demonstrate a convex soft tissue shadow in contact with the posterior surface of the sternum. The hematoma in the case herein reported was most prominent in the posteroanterior view but was also clearly visualized on the lateral x-ray.

X-ray diagnosis of traumatic mediastinal hematoma in the absence of a history or roentgen evidence of gross chest injury is necessarily tenuous. On the other hand, a history of trauma should not be permitted to exclude the possibility of an underlying mediastinal neoplasm. The tendency of traumatic mediastinal hematomas to progressive and usually rapid resolution militates against diagnostic temporizing in cases which exhibit little change.

As in the patient described, conservative treatment of traumatic mediastinal hematoma is usually successful. However, especially in the early phase, signs of significant cardiorespiratory embarrassment must be promptly detected and actively treated. Decompression of the mediastinum, either by needle aspiration or by anterior mediastinotomy, may be required. In arterial hemorrhage, operative ligation of the bleeding vessels may be necessitated for secure hemostasis. Associated injuries and complications must, of course, be adequately managed.

SUMMARY

Hemomediastinum is a striking condition which may complicate the course of numerous diseases or may be associated with severe chest trauma. Its incidence following thoracic injury is believed to be higher than indicated by the literature. A case of traumatic mediastinal hematoma with successful conservative management is presented. An interesting clinical finding was the presence of a transient unilateral Horner's syndrome. The source of bleeding in traumatic hemomediastinum is usually venous but may be arterial if the injury is penetrating. Physical findings are dependent upon the extent, location, and rapidity of accumulation of the hematoma. X-ray characteristics are those of enlargement of the supracardiac shadow with straight, smooth, sharply defined margins. Conservative treatment is usually adequate although severe cardiorespiratory embarrassment may require mediastinal decompression.

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STUDIES ON FIBRINOLYTIC ENZYMES IN PATIENTS UNDERGOING THORACIC SURGERY

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IN RECENT years uncontrollable hemorrhage following thoracic operations has been reported.[1-4] In these cases the bleeding was diffuse, did not originate in major open vessels, and was ascribed to afibrinogenemia due to fibrinolysis.

During the three-year period ending July 1, 1954, 342 major thoracic interventions such as pulmonary resection, thoracoplasty, and cardiac operations were performed on our service. In this series two severe hemorrhages occurred, both after pulmonary resection. In one case a ligature had slipped off the pulmonary artery, and in the other, bleeding was due to erosion of an intercostal artery.

In the present communication, studies are reported on fibrinolytic enzymes in the blood before, during, and after operation and in blood obtained from the open chest during operation in patients undergoing major thoracic surgery.

METHODS

Blood samples were obtained from the brachial vein, the pulmonary artery, the left atrium, and the open chest. No saline or wet sponges were used until the pleural blood samples had been taken in order to avoid dilution. The samples obtained with or without anticoagulant (⅑ volume of 0.1 M sodium oxalate) were centrifuged at 2,000 r.p.m. for 15 minutes. The plasma or serum was separated and kept at +4° C. for a maximum of 3 hours until the performance of the tests.

The following materials were used:

Barbital saline buffer, pH 7.4 : 11.0 Gm. NaCl, 8.244 Gm. sodium barbital and 0.924 Gm. potassium oxalate dissolved in 2 L. of distilled water.

Fibrinogen: a 600 mg. per cent Bovine Fraction I (Armour Co.) solution in barbital saline buffer, freshly prepared. The material contained profibrinolysin.

Thrombin (Upjohn Co.): dissolved in saline barbital buffer to a strength of 50 units per milliliter.

Profibrinolysin: prepared from human serum by the method of Ratnoff.[5]

Streptokinase-streptodornase—Varidase (Lederle Co.): dissolved in barbital saline buffer to a strength of 1,000 units per milliliter.

Fibrinolysin: a bovine preparation received through the courtesy of Dr. E. C. G. Loomis from Parke, Davis and Co.

Fibrin plate: prepared according to Astrup and Müllertz,[6] heated to 85° C. for 45 minutes in order to destroy profibrinolysin.

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Tests for the presence of the following substances were performed:

Fibrinogen: to 0.5 ml. test sample mixed with 0.4 ml. buffer solution, 0.1 ml. thrombin solution was added at 37° C. and the mixture was examined for the appearance of a clot—*method A.*

Fibrinolysin: to a mixture of 0.5 ml. test sample and 0.4 ml. fibrinogen solution, 0.1 ml. thrombin solution was added. The clot was observed for lysis at 37° C. during a period of 48 hours—*method B.*

A similar test was performed on a 1,024-times diluted sample in order to abolish antifibrinolysin activity—*method C.*

In a third method 0.05 ml. of the test sample was placed on a heated fibrin plate which was observed for the appearance of a halo at 37° C. for a period of 48 hours—*method D.*

Profibrinolysin: to a mixture of 0.05 ml. test sample, 0.05 ml. streptokinase solution, and 0.4 ml. fibrinogen solution, 0.1 ml. thrombin solution was added, and the clot was observed for lysis at 37° C., during a period of 48 hours—*method E.*

Profibrinolysin activator (fibrinolysokinase): 0.1 ml. of a mixture containing equal parts of test sample and profibrinolysin solution was placed upon a heated fibrin plate which was kept at 37° C. The appearance of a halo within 48 hours indicated the presence of activator—*method F.*

Antifibrinolysin: titrated according to Ratnoff[7]—*method G.*

RESULTS

In the 11 cases studied, the operative and postoperative courses were uneventful, and no undue bleeding was observed. The results of the laboratory studies in these 11 cases are presented in Table I.

In all blood samples obtained from the brachial vein, the pulmonary artery, and the left atrium, adequate clots appeared within a few seconds after the addition of thrombin. In none of these samples could fibrinolytic activity be demonstrated by the different methods used. In all samples examined, profibrinolysin was present and fibrinolysokinase was absent. Antifibrinolysin titers in the blood samples from the peripheral and intrathoracic vessels did not show marked changes during operation.

All blood samples obtained from the open chest either contained clots or clotted in the test tube. In none of these samples was fibrinolysin or fibrinolysokinase demonstrated, but all contained profibrinolysin. Antifibrinolysin titers in 9 of the 10 examined pleural blood samples were markedly lower than in the samples obtained simultaneously from the peripheral or intrathoracic blood vessels. Only in one sample (Case 7), which was removed from the open chest before it clotted, was the serum antifibrinolysin titer increased as compared with the titer in the peripheral blood.

COMMENT

Lung tissue is known to be rich in thromboplastin[8] and tissue fibrinolysokinase.[9] Afibrinogenemia or hypofibrinogenemia reported to occur in thoracic surgery might be explained by one of the following mechanisms: (1) intra-

TABLE I. STUDIES OF FIBRINOLYTIC ENZYMES IN BLOOD OBTAINED FROM THE PERIPHERAL AND LESSER CIRCULATION AND FROM THE OPEN CHEST IN THORACIC OPERATIONS

	ORIGIN OF BLOOD SAMPLES	PRESENCE OF CLOTS	FIBRINOGEN METHOD A	FIBRINOLYSIN METHOD B	C	D	PROFIBRINOLYSIN METHOD E	FIBRINOLYSOKINASE METHOD F	ANTIFIBRINOLYSIN TITER, METHOD G CASE 1	2	3	4	5	6	7
LUNG SURGERY 7 Cases	*Open Chest*														
	Before lung resection	(7)+	(6)–, (1)+*	(7)–	(7)–	(4)–	(7)+	(7)–	15	30	50	100	100	96	800
	After lung resection	(7)+	(6)–, (1)+*	(7)–	(7)–	(4)–	(7)+	(7)–							
	Brachial Vein														
	Before operation		(7)+	(7)–		(5)–	(7)+	(5)–	280	150	300	500	600	360	800
	During operation		(3)+	(3)–		(3)–	(3)+	(2)–	300						800
	At end of operation		(4)+	(4)–			(4)+	(4)–	300		300	450		360	800
	Twenty-four hours after operation		(2)+	(2)–			(2)+	(2)–	300	150					

									CASE 8	9	10
HEART SURGERY 4 Cases	*Open Chest*										
	Before intracardiac procedure	(3)+	(3)–	(3)–	(3)–		(3)+	(3)–	20	50	50
	After intracardiac procedure	(3)+	(3)–	(3)–	(3)–		(3)+	(3)–			
	Pulmonary Artery										
	During operation		(4)+	(4)–			(4)+	(4)–	600		300
	Left Atrium										
	During operation		(4)+	(4)–			(4)+	(4)–	600		300
	Brachial Vein										
	Before operation		(3)+	(3)–			(3)+	(3)–	550	350	300
	At end of operation		(2)+	(2)–			(2)+	(2)–	450	320	225

Numbers between parentheses indicate the number of cases examined.
*Blood obtained from thorax before clotting; clotted in test tube.

vascular clotting by thromboplastin released from the lung tissue into the circulation; (2) activation of plasma profibrinolysin to fibrinolysin by tissue kinase from the lungs[9]; and (3) release of active fibrinolysin from the tissues into the circulating blood.[10]

In our 11 cases, severe hypofibrinogenemia did not occur. Also no active fibrinolysin or fibrinolysin activator could be demonstrated in the blood. Even blood samples obtained from the open chest, which had been in contact with traumatized tissue, did not show fibrinolytic activity. A remarkable as yet unexplained finding is the low antifibrinolysin titer in the pleural blood samples as compared to those obtained by puncture of blood vessels. The decrease in antifibrinolysin titer in these samples did not seem to have clinical significance since fibrinolytic activity or undue bleeding did not ensue.

It follows, therefore, that the appearance of fibrinolytic activity in the peripheral and the pulmonary circulation in thoracic surgery is not a common occurrence. This is in consonance with the absence of undue diffuse bleeding in our surgical material. When hypofibrinogenemia does occur in thoracic operations, an additional, so far unknown mechanism must be involved.

SUMMARY

In patients undergoing lung resection or intracardiac surgery and who had no undue diffuse bleeding or marked hypofibrinogenemia, fibrinolysin and fibrinolysokinase could not be demonstrated in blood from the peripheral veins, the pulmonary artery, and the left atrium, while profibrinolysin was present.

Blood obtained from the open chest contained clots and did not show fibrinolysin or fibrinolysokinase activity but had markedly lowered antifibrinolysin titer.

Afibrinogenemia appears to be uncommon in thoracic surgical patients, and the mechanism of its occurrence is not understood.

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USE OF THE LITTLE FINGER FOR MITRAL COMMISSUROTOMY

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IN THE MAJORITY of patients with uncomplicated mitral stenosis, the operation of mitral commissurotomy is a technically straightforward and effective procedure, as is attested to by the low operative risk and favorable postoperative improvements reported for these patients by many surgeons in widely differing geographic areas.

In the occasional patient in whom the left auricular appendage is too small to admit the forefinger, however, the morbidity and mortality of the operation are significantly higher. In such a case one must either forego exploring the heart (which hardly seems an adequate solution to the problem) or approach the diseased valve through the left atrial wall proper or through one of the left pulmonary veins. In our own experience, each of these latter two approaches has been associated with significantly greater and occasionally formidable technical difficulties of the nature which have been described by Bailey[1] and which account for the greater risk and less favorable results when these approaches are utilized by necessity.

Recently we have encountered two such patients in whom the auricular appendages would not come close to admitting the operator's forefinger, but were of sufficient size to admit the fifth finger. In both cases the commissurotomy was accomplished with surprising ease and a gratifying effectiveness—so much so that we believe it of value to call to the attention of other surgeons the value of this simple maneuver.

At first thought, one might consider the use of the little finger like sending a "boy to do a man's job"; however, when one considers the fact that the fifth finger may be rigidly fixed in a semiflexed position and that the whole hand and arm may then be utilized to lift the fifth finger, one sees that surprising leverage can be exerted to split the anterior commissure. Moreover, the shorter length of the fifth finger as compared to the forefinger is not as great a handicap as one might surmise because inserting the shorter fifth finger through the left auricular appendage actually puts one in a position appreciably closer to the mitral valve than can be obtained by introducing the longer forefinger through the more distant superior pulmonary vein. This observation is dependent upon the fact that in the average individual the

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difference in length between the forefinger and the fifth finger is less than 1.5 cm., whereas the left auricular appendage is 3 to 5 cm. or more closer to the mitral valve as compared to the left superior pulmonary vein.

Also, if it is deemed necessary, one may further decrease the diameter of the finger and take advantage of the sharper fingernail edge by removing the rubber glove from this finger.

The following two case reports illustrate the application of this technique for commissurotomy.

A. B. (U.H. No. 871612), a 47-year-old man, was admitted to the University Hospitals on June 7, 1954. This patient was first known to have cardiac difficulty six years prior to admission when a routine chest x-ray disclosed an enlarged heart. He had no history suggestive of rheumatic fever. Shortly thereafter he was hospitalized for decompensation and treated with digitalis, diuretics, and low salt diet. He had continued daily digitalis and intermittant diuretics with no further bouts of decompensation. For the two years preceding this admission he has had progressive exertional dyspnea such that he was unable to be gainfully employed.

Physical examination revealed an enlarged heart, rumbling diastolic murmur at the apex, accentuated P_2, and auricular fibrillation. He was well compensated without evidence of pulmonary or peripheral edema or an enlarged liver.

Surgery was undertaken on June 10, 1954, utilizing a left lateral approach through the bed of the fourth rib. A diastolic thrill was palpable at the upper border of the left ventricle. After opening the pericardium, the left auricular appendage was noted to be very small, and it was doubtful that it could be used to enter the left atrium. A purse-string suture was placed about its base, however, and the appendage was opened and an attempt to insert the forefinger was made without success.

We were about to tie off the appendage and approach via the left atrial wall proper or pulmonary vein when it was suggested that an attempt be made to insert the little finger. This was subsequently done, and exploration disclosed a stenotic valve with an orifice of 5 mm. The anterior commissures were widely split with ease, using the fifth finger. The result was most gratifying, and the patient had an uneventful postoperative convalescence.

I. H. (U.H. No. 870863), a 48-year-old white woman, was admitted to the University Hospitals on June 2, 1954, with a history of rheumatic fever at 14 years of age and with the onset of exertional dyspnea 15 to 20 years prior to this admission. She had first become decompensated nine years previously and had had intermittent bouts of failure with ascites, hepatomegaly, and persistent auricular fibrillation despite digitalis, low salt diet, and the occasional use of mercurial diuretics.

Physical examination revealed evidence of cardiac enlargement, mid-diastolic rumbling murmur at the apex, auricular fibrillation, enlarged liver, 2+ pitting edema of both ankles, and occasional râles at both lung bases.

Electrocardiogram revealed right axis deviation and atrial fibrillation. Cardiac fluoroscopy showed right ventricular and left atrial enlargement.

With the use of mercurial diuretics she subsequently lost 10 pounds of weight. The basilar râles and ankle edema disappeared, and she was taken to the operating room for mitral valve surgery on June 18, 1954. A diastolic thrill was palpable over the upper border of the left ventricle and a systolic thrust over the left atrium. The auricular appendage was small, and again it appeared doubtful whether it would accept the forefinger. Moreover, an attempt to enter the atrium by means of the forefinger through this appendage was unsuccessful even with the glove removed from the finger. In addition there was noted an organized thrombus on the adjacent left auricular wall. Because of the previous experience in the above-mentioned case, the fifth finger was utilized, with the glove over this finger cut off. This finger

entered the atrial chamber without difficulty. Exploration disclosed a thick stenotic valve with a slight amount of insufficiency posteriorly. The considerable degree of stenosis present at the anterior commissures was easily split open and without any evidence of increased insufficiency. This patient also has had an uneventful postoperative convalescence.

DISCUSSION

It is, perhaps, obvious that one may still encounter an occasional patient in whom the left auricular appendage may not admit even the little finger. In such patients one must be prepared to utilize one of the other alternatives that have been mentioned. It is our belief, however, that the use of the fifth finger in patients with very small left auricular appendages will make the need for these more hazardous approaches infrequent.

SUMMARY

One of the problems encountered in mitral valve surgery is the occasional patient with a very small left auricular appendage. In certain of these patients the use of the little finger (with or without the glove removed) introduced through the left auricular appendage allows one to perform mitral commissurotomy with greater ease and safety than an approach through either the left atrial wall or pulmonary veins.

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SUBVALVULAR MITRAL STENOSIS—ITS RECOGNITION AND TREATMENT

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IN THE performance of mitral commissurotomy for mitral stenosis, it has become more and more apparent to us that, in order to relieve the stenosis as completely as possible and also to avoid the production of mitral insufficiency, one must be extremely cognizant of the subvalvular chordopapillary supporting structures. We have elected to use the term "subvalvular stenosis" to indicate the presence of any pathologic alteration on the ventricular side of the mitral leaflets which impedes blood flow through the atrioventricular channel. This condition is caused by fusion of the papillary muscles and/or chordae tendineae on the ventricular side of the valve. It can occur with or without marked shortening of the chordae tendineae.

We have attempted to divide cases of subvalvular stenosis into three types for descriptive purposes and also for recognition and surgical alleviation at the time of operation.

In type one, the chordae tendineae are cross-fused and give the tactile impression of a bird-cage or netlike structure blocking the flow of blood (Fig. 1, *B*). In this type, finger dissection alone has proved to be the most satisfactory solution in our experience. The finger pressure is applied downward in a ventricular direction to separate the fused tendinous groups, those to the mural and septal leaflets, respectively (Fig. 1, *C*). If the chordae are not completely fused all the way to the papillary muscle, one can, at times, insert his finger between the two groups of chordae tendineae at the tip of the papillary muscle and apply pressure upward toward the atrium to get resultant splitting (Fig. 1, *D*). If neither of these methods is satisfactory, one can use the finger to perforate the network by downward pressure and clear a passageway for atrioventricular blood flow. This may result in minor degrees of regurgitation, but it will remain minor so long as the major chordae to the leaflets are not broken.

In type two, there is chordo-papillary fusion and marked shortening of the chordae tendineae, in some cases to a length of 0.5 cm. On digital examination, one can feel the nearness of the papillary muscles to the leaflets and can feel the Y-shaped spreading of the chordae tendineae as they leave the tip of the muscle to their insertion into the respective leaflets (Fig. 2, *A*).

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The distance between the papillary muscle and the fused valve edges is so small, however, that the standard guillotine knife, developed by Bailey, Glover, and O'Neill, cannot be easily engaged into the space. We have devised a guillotine knife which has been most advantageous in coping with this type of subvalvular stenosis (Fig. 3). The lower end of this knife is blunt and probelike and presents a golf-stick-like angle at its point of take-off from the

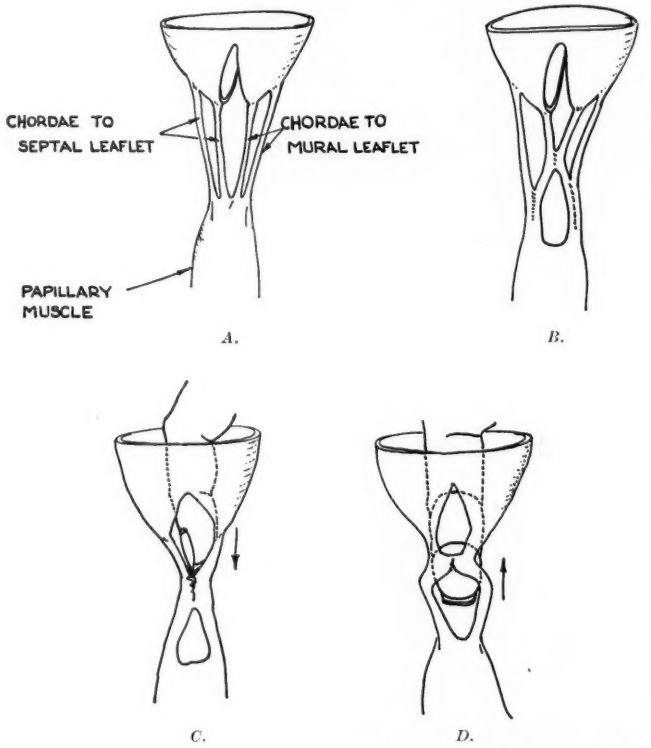

Fig. 1.—*A*, Normal chordae and papillary muscle support to mitral valve. *B*, Cross-fusion of chordae in type one subvalvular stenosis. *C*, Finger pressure from above to free type one stenosis. *D*, Finger pressure from below to free type one stenosis.

shaft of the instrument. This lower probelike end protrudes beyond the guillotine blade when the knife is closed, but, being blunt, the closed knife has no presenting sharp edges. The blunt protruding lower end is used to seek out the passage between the papillary muscle and the mitral leaflets (Fig. 2, *B*). After it has been engaged into this space, the instrument is then moved from side to side toward the mural leaflet and septal leaflet, respectively, to be sure that it is engaged so as to have chordae tendineae on both sides. Since there is no cutting surface on the closed instrument, several probing passes can be made with it without fear of injuring the chordae tendineae. The

engaged lower edge can be made to protrude upward against the valve so that it can be palpated by the index finger on the atrial surface of the commissure to ensure that the proper commissural alignment has been made (Fig. 2, *C*).

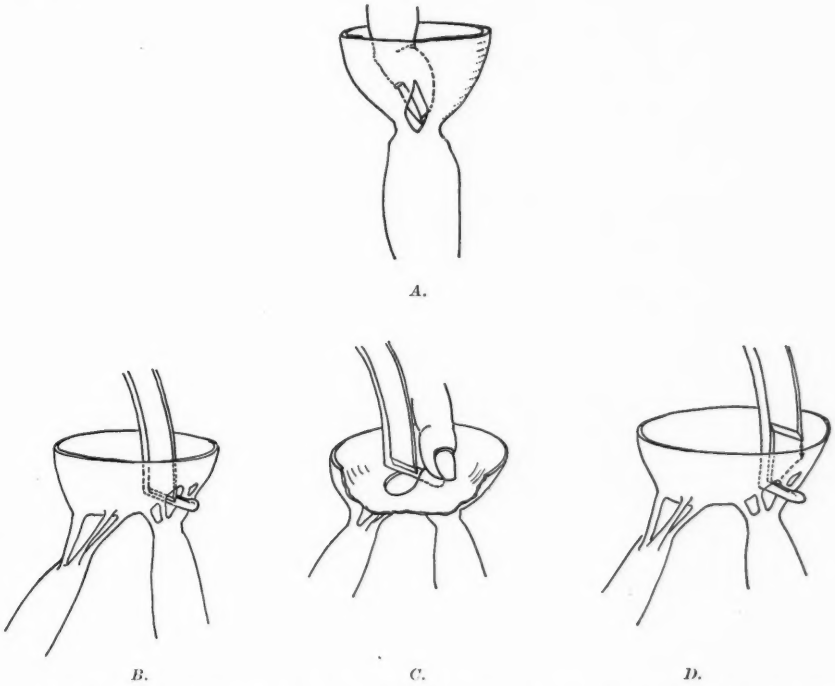

Fig. 2.—*A*, Palpation of Y-shaped fusion of thickened and shortened chordae in type two subvalvular stenosis. Papillary muscle near leaflet edges. *B*, Probe end of closed guillotine knife engaged into small Y-shaped area beneath fused commissure. *C*, Finger palpation on the atrial surface of the commissure to ensure proper alignment of knife. *D*, Opened knife is fully engaged and ready for guillotine cutting of commissure.

Next, the guillotine is opened, and the knife is engaged deeper into the commissure. The guillotine is then closed and proper commissural cutting is accomplished (Fig. 2, *D*). It should be noted that the guillotine blade of this instrument is slightly narrower than that of the standard guillotine knife so as to ensure a smaller cut. In many instances, one small cut is all that will be necessary as the remaining commissural fusion may be split by digital pressure against the apex of the incision. If further cutting is necessary, it is better to palpate the result of the previous cut and be certain of the anatomy of the remaining stenosis before again engaging the knife to carry out opening of the commissure all the way to the atrioventricular ring. The multiple cut method is of the most importance when there is an exaggerated elliptical shape to the fused commissure. With multiple small cuts, the natural curve of the line of

valve closure can be followed safely to the atrioventricular ring, whereas a single large cut might extend peripherally into one of the leaflets to produce resultant regurgitation.

After the commissure has been opened, digital pressure downward into the fork of the Y-shaped chordal attachment to the papillary muscle will often result in papillary muscle splitting which will result in greater freedom of the leaflet edges to swing widely apart during diastole. If the papillary muscle does not respond to digital splitting, a Papillotome may be used to incise the muscle. This instrument and its application will be described in the treatment of the type three stenosis.

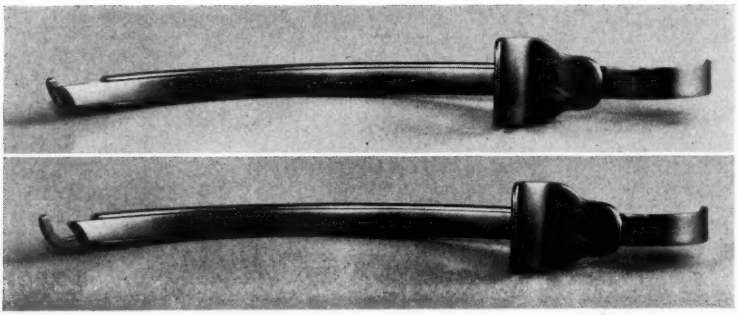

Fig. 3.—Guillotine knife with blunt, golfstick-angled lower end. (Mfg. by George P. Pilling and Son, Philadelphia, Pa.)

In type three, there is leaflet-papillary fusion and marked shortening of the chordae tendineae to the point that the latter are not actually identifiable as such to the palpating finger. There is actually fusion of the papillary muscles to the edges of the mitral leaflets (Fig. 4, *A*). This type of valve is recognized when one passes his finger through the mitral orifice and extends it out beneath the commissure on the ventricular side of the valve. One then feels a thickened, fleshy bar present, but no identification of chordae tendineae can be made. A slight variation of this is the palpation of the papillary muscle mass, fusing to the mural leaflet edge, and the absence of any palpable chordae tendineae or papillary muscle to the septal leaflet edge. In this condition, the chordae from the septal leaflet are agglutinated to the ventricular surface of the fused commissure on their course to the papillary muscle.

It has been our experience that the standard guillotine knife has not been entirely satisfactory in relieving this type of subvalvular stenosis. Since the hook on the knife points back up the shaft of the instrument, it cannot be easily engaged into the papillary muscle mass so as to ensure division of this mass along with the fused commissure. It is of prime importance that the papillary muscle mass be so divided as to ensure maintenance of support to both mitral leaflets.

We have devised a guillotine knife, the "Papillotome" (Fig. 5), in which the lower end has been made straight instead of containing a hook. Also, this lower end has been made to angulate from the instrument, in "golfstick" fashion so its sharp lancet point can be plunged into the papillary muscle mass perpendicular to its presenting surface. This guillotine knife, when closed,

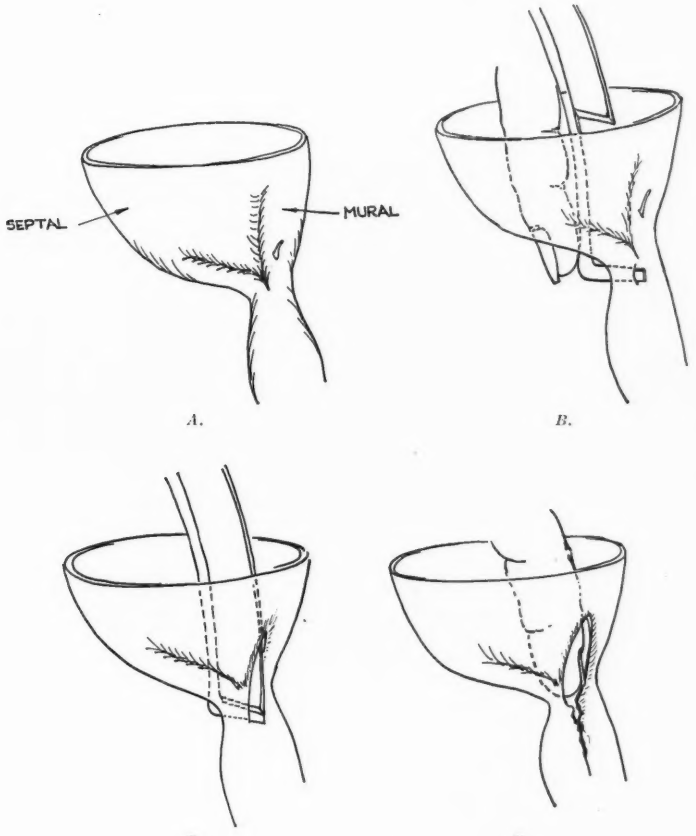

Fig. 4.—*A*, Type three subvalvular stenosis where chordae are so short and thick that they cannot be definitely identified. To the palpating finger the papillary muscles seem to attach directly to the leaflet. *B*, The lancet end of the Papillotome is inserted perpendicularly into the central area of the papillary muscle mass. *C*, Guillotine cutting starts at the commissure on the atrial surface and extends down into the papillary muscle to the original point of Papillotome insertion. *D*, Digital pressure to produce further splitting of the papillary muscle.

presents no cutting edges, so that it can be safely introduced into and withdrawn from the heart. After the knife has been introduced into the left atrium, along the right index finger, the guillotine is opened and the lancet is inserted into the papillary muscle mass (Fig. 4, *B*). The guillotine is then closed, and

the cutting action starts on the atrial surface of the fused commissure and extends on down into the center of the papillary muscle mass to the point where the lancet was originally engaged (Fig. 4, *C*). By this method the support to each leaflet is maintained by the respective half of the divided muscle. Digital pressure downward into the apex of the incision into the papillary muscle will usually result in further splitting which allows the mitral leaflet edges to fall widely apart during diastole (Fig. 4, *D*). If the digital pressure is not adequate because of dense fibrosis, another cut with the knife may be necessary.

DISCUSSION

The first important aspect of subvalvular stenosis is the recognition of its presence at the time of mitral commissurotomy. This recognition is greatly enhanced in our experience since we have adopted the bare-finger technique. The increased tactile sense of bare finger over gloved finger can be appreciated only after it has been employed in an operative case. If the subvalvular stenosis is not recognized, the chordae tendineae and papillary muscles can easily be cut at the time the fused commissure is being incised.

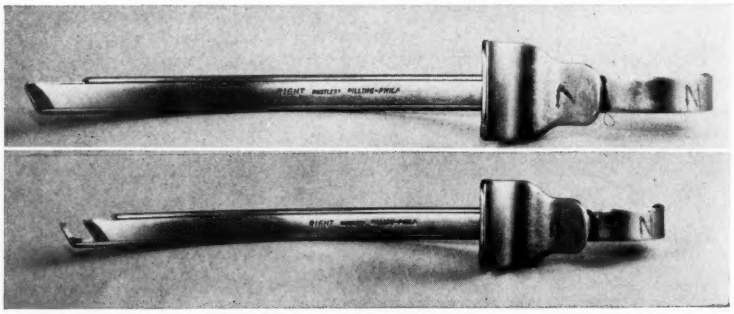

Fig. 5.—Papillotome. (Mfg. by George P. Pilling and Son, Philadelphia, Pa.)

Such cutting of these subvalvular structures removes the support to the valve edges and invariably produces regurgitation. This might well result in an immediate or delayed fatality or, at best, a poor clinical result.

The second important aspect to be considered is the freeing up of the subvalvular stenosis to allow a maximum of atrioventricular blood flow. Separation of fused chordae and of fused papillary muscles is essential to allow the leaflet edges to fall widely apart during diastole. In the type three stenosis described, even though the papillary muscle is deeply split or incised as suggested, there is still some impedance to atrioventricular blood flow as the blood has to spill over the bulky papillary muscle mass, whereas in a normal heart, it would have only to course around the fine and delicate chordae to enter the left ventricular chamber. We have no suggestion as to how this final bit of subvalvular obstruction can be overcome.

SUMMARY

Subvalvular stenosis is being recognized with increasing frequency during mitral commissurotomy. Three types of subvalvular stenosis have been recognized by the ungloved palpating finger of the operator. The technique of treating each of the three types has been described. Two types of guillotine knives which have greatly augmented the successful operative technique are described for the first time.

SURGICAL TREATMENT OF MITRAL INSUFFICIENCY

Experimental Observations

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RHEUMATIC involvement of the valvular components of the mitral valve may result in mitral stenosis, a combination of stenosis and regurgitation, and what is termed "pure mitral regurgitation." Initially, there is absolute loss of valve substance by cicatricial contraction of the leaflets which prevents coaptation of the valve cusps during ventricular systole. This may or may not be associated with retraction of the valve cusps into the ventricular chamber by scarred and contracted chordae tendineae. The resultant regurgitation of blood during ventricular systole causes the left ventricle to hypertrophy and dilate. There is a delicate balance in this compensatory mechanism between the regurgitation and the ventricular hypertrophy. If there is chronic stress on the heart to increase cardiac output, sufficient ventricular hypertrophy and dilatation may result to cause dilatation of the atrioventricular ring which, in turn, causes further separation of the valve cusps. This relative loss of valve substance as a result of dilatation of the mitral ring now becomes one of the most important factors in the consideration of its surgical correction.

Examination of the insufficient valve with the intracardiac finger reveals that the regurgitant flow is entirely through the posteromedial one-half of the mitral orifice. The anterior cusp of the mitral valve forms part of the aortic outflow tract during ventricular systole. The absolute, as well as relative, reduction in valve substance from the cicatricial contraction of the leaflets, with or without retraction of the leaflets into the ventricular chamber by the contracted chordae tendineae, and separation of the posteromedial commissure from dilatation of the mitral ring, gives rise to a "tear-drop" type of deformity with loss of occluding valve substance in the medioposterior one-half of the orifice which allows the jet of regurgitant blood to be directed posteriorly toward the atrial septum.

Previous techniques at surgical repair of mitral insufficiency have attempted to substitute various types of grafts[1, 2] for valve substance or to occlude the incompetent opening by various types of plastic baffles.[3, 4] This latter technique has the obvious disadvantage of introducing a foreign body in the heart. We have had initial success with the use of pericardial grafts only to have the graft become fibrotic and ineffective at a later date. This experience with the use of a pericardial graft has been noted by others. Davila and associates[5] recently reported experimental observations relative to reducing the size of the mitral ring by a circumferential suture about the ring.

The regurgitant flow of blood can be entirely stopped, as determined by the intracardiac finger, by pushing the posterior wall of the auricle at the site of the mitral valve forward or anteriorly, thereby decreasing the size of the

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mitral ring in an anteroposterior direction. With this concept in mind, anatomic dissections and laboratory experiments were performed in an attempt to devise a technique which would reduce the size of the mitral ring at the posteromedial commissure which, in turn, would approximate the edges of the valve leaflets and obliterate this incompetent space.

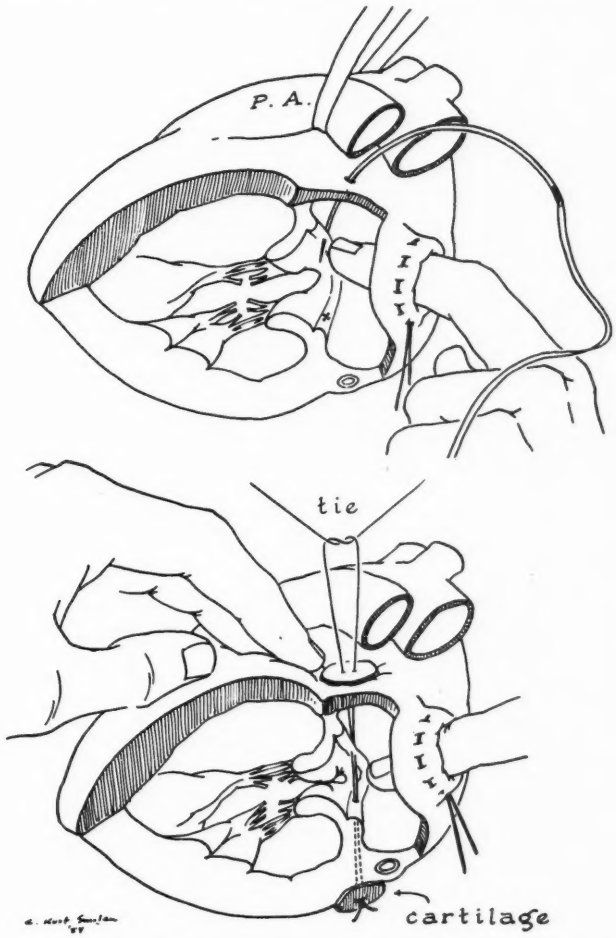

Fig. 1.—Diagrammatic sketches of the technique of introducing sutures to reduce the size of the enlarged mitral annulus for the correction of mitral regurgitation.

Anatomic dissection of normal hearts and of patients dying of mitral insufficiency showed the incompetence could be relieved by introducing a double suture at the junction of the aorta and auricles, through the anterior and posterior segments of the mitral ring adjacent to the posteromedial commissure and out the ventricular side of the coronary sinus in the region free of the circumflex coronary artery posteriorly (Fig. 1).

In the experimental animal this double suture is drawn taut, approximating the anterior and posterior segments of the mitral ring as determined by the intracardiac finger, and tied over sections of cartilage. The surfaces of the dilated ring should be approximated so no suspended suture lies free within the auricular chamber. The technique consists of retracting the aorta and pulmonary artery anteriorly. The index finger of the left hand is introduced into the left auricle through the auricular appendage. The desired site anterior and posterior to the separated posteromedial commissure for placing the suture is palpated. A large blunt-tipped, curved needle is introduced into the auricular chamber at the junction of the aorta and auricles adjacent to the auricular septum. The tip of the needle is guided by the intracardiac finger to engage first the anterior margin and then the posterior margin of the valve ring, and to exit on the ventricular side of the coronary sinus in the area just distal to the circumflex coronary artery. A double suture of No. 1 silk is pulled through. The suture is then threaded through sections of cartilage and drawn taut and tied.

Clinically, one or two sutures have sufficed. Also, clinically, it has been easier upon occasion, because of the enlarged left auricle, to introduce the needle through the opening in the auricular appendage, through the anterior segment of valve ring, and out at the junction of the aorta and auricles. The other end of the suture is then threaded and passed in a similar manner through the opening in the auricular appendage, the posterior margin of the valve, and out posteriorly. The two free ends are tied and pulled through as a double suture. The clinical application of this technique will be published later.

This technique corrects two of the three factors responsible for the insufficiency. It reduces the size of the dilated mitral ring and compensates for the loss of valve substance by approximating the edges of the valve leaflets.

Fixation of the valve leaflets in the open position by contracted chordae tendineae, if present, is partially corrected by severing the more foreshortened of these by means of a right-angled neurectomy knife inserted into the left ventricular chamber and guided by the intracardiac finger. The use of these two techniques appears to be satisfactory in the clinical correction of mitral insufficiency, the details of which are to be published.

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TRAUMATIC PNEUMOTHORAX WITHOUT RIB FRACTURE

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THE literature has very few case reports of traumatic pneumothorax without rib fracture, so we felt it would be well to report two such cases with a discussion of traumatic pneumothorax and the possible mechanism involved in this type of injury.

Traumatic pneumothorax is secondary to external violence, with or without an opening in the chest wall, resulting in rupture of the nondiseased visceral pleura, lung, or bronchi, with escape and accumulation of air in the pleural cavity.[1]

The presence of air in the pleural cavities was recognized in the Hippocratic writings under the term empyema, but the condition was not understood. Meckel, in 1759, first clearly recognized the presence of air in the pleural space and understood the effect on respiration.[2] Itard, in 1803, gave pneumothorax its name, and his work along with that of Laennec, in 1819, established pneumothorax clinically.[3] Saussier, in 1841, divided pneumothorax etiologically into traumatic and nontraumatic pneumothorax.[4]

Dieffenbach,[5] in 1926, reported two cases of lung rupture without any evidence of external injury or rib fracture. He made the observation that such injuries were more often found in children, in whom the chest wall is still very pliable.

Sealy,[6] in 1949, reported 40 cases of contusions of the lung from nonpenetrating injuries to the thorax. He commented that it was unusual that one case had hemopneumothorax without evidence of rib fracture.

CASE REPORTS

CASE 1.—L. F., a 3½-year-old white child, was admitted to St. Joseph Hospital, Sept. 3, 1950, because he had pain in the abdomen. The patient had been struck by an automobile one week prior to admission, but there was no external evidence of injury to the chest. Following the accident the patient was brought to the Emergency Room of St. Joseph Hospital, where superficial abrasions were treated, and the patient was allowed to go home. Shortly after the accident the child began to appear short of breath and developed a cough and "tight chest." Several days prior to admission he began to have fever and complain of right upper abdominal pain.

Physical examination revealed a well-developed, well-nourished, acutely ill child whose temperature was 103° F.

Examination of the chest showed the right side did not expand as well as the left. The right chest was hyperresonant to percussion with dullness to percussion in the right base. Tactile and vocal fremitus were decreased on the right. The trachea and mediastinum were shifted to the left. The remainder of the physical examination revealed no abnormalities.

Laboratory examinations revealed a red blood cell count of 4,040,000 per cubic millimeter, hemoglobin 11.6 Gm. per 100 c.c., and leukocyte count 16,300 per cubic millimeter

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with 73 per cent segmenters, 5 band forms, and 22 per cent lymphocytes. Urinalysis yielded a negative sugar and protein, 4 plus acetone, and an occasional leukocyte per microscopic high-power field.

Roentgenograms of the chest exhibited a complete collapse of the right lung with a fluid level at the base. There was no evidence of rib fracture (Fig. 1).

Under 1 per cent procaine infiltration anesthesia, an intercostal catheter was inserted into the right third intercostal space in the mammary line and attached to a water-seal drainage bottle. A large amount of air was evacuated from the pleural space by this method. Aqueous penicillin, 100,000 units, and streptomycin, 0.5 Gm. in 10 c.c. of saline, were placed in the right pleural space.

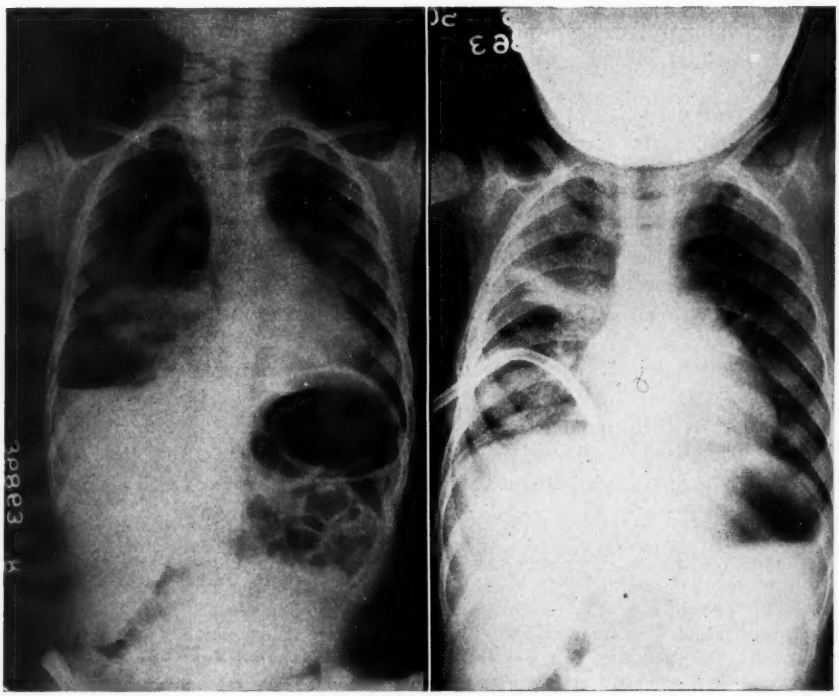

Fig. 1.—Roentgenograms show complete collapse of the right lung with a fluid level at the base and complete re-expansion with intercostal catheter in place.

The lung had completely re-expanded in two days and the intercostal catheter was removed. The child has remained well since leaving the hospital.

CASE 2.—R. L., a 42-year-old white man, was admitted to the Good Samaritan Hospital June 26, 1951, because he had been in an automobile accident just prior to admission. He had apparently been in good health before the accident.

Physical examination revealed a well-developed, well-nourished, acutely ill man who was moderately cyanotic and was having some respiratory difficulty. There were obvious fractures of the right clavicle, right tibia, and left femur.

Examination of the chest showed the left side did not expand as well as the right. The left chest was hyperresonant to percussion. Breath sounds were decreased on the left side. The trachea and mediastinum were shifted to the right.

Pulse rate was 85 with sinus rhythm. Systolic blood pressure was 120 mm. Hg, and diastolic pressure 70 mm. Hg. The heart was not enlarged and no murmurs were audible. The remainder of the physical examination revealed no abnormalities.

Laboratory examinations revealed a red blood count of 4,490,000 per cubic millimeter, hemoglobin 12.2 Gm. per 100 c.c., and leukocyte count 12,500 per cubic millimeter with 81 per cent segmenters and 19 per cent lymphocytes. Urinalysis yielded a specific gravity of 1.023, no sugar, 1 plus protein, 2 to 3 leukocytes per microscopic high-power field, and 1 to 3 granular casts per microscopic high-power field.

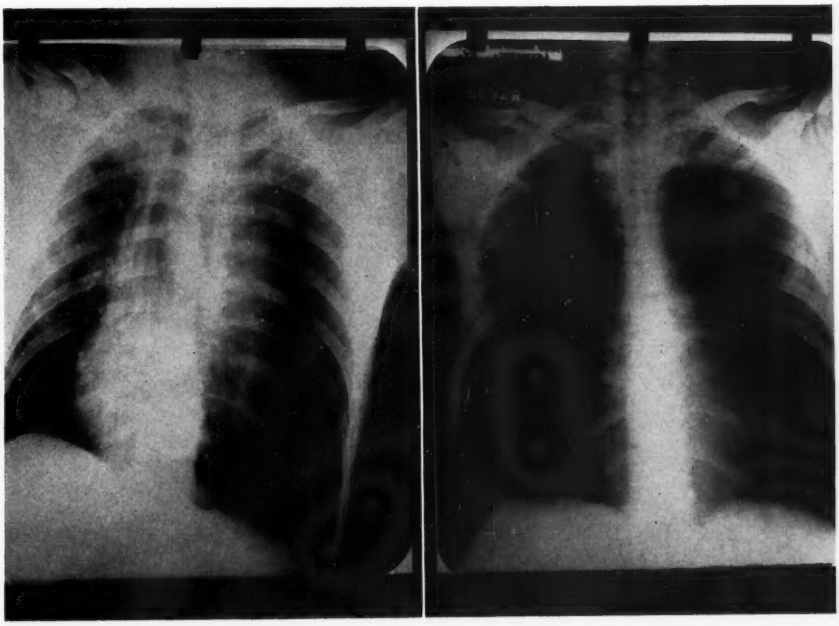

Fig. 2.—Roentgenograms show evidence of a pneumothorax involving the left thoracic cavity with no evidence of rib fracture and complete re-expansion after adequate therapy.

Roentgenograms of the chest exhibited evidence of a pneumothorax involving the left thoracic cavity, with the lung being about 80 per cent collapsed. The heart and mediastinum were displaced markedly toward the right and the diaphragm was displaced downward slightly. No evidence of rib fracture was present (Fig. 2).

Under 1 per cent procaine infiltration anesthesia, an intercostal catheter was inserted into the left fourth intercostal space in the anterior axillary line and attached to a water-seal drainage bottle. A large amount of air was evacuated from the pleural space by this method. Aqueous penicillin, 100,000 units, and streptomycin, 0.5 Gm. in 15 c.c. of saline, were placed in the left pleural space.

The lung had completely re-expanded in four days and the intercostal catheter was removed. The lung has remained expanded and there have been no subsequent pulmonary symptoms.

DISCUSSION

The etiology of traumatic pneumothorax without rib fracture has not been proved to our knowledge. Lindskog and Liebow[7] have stated that nonpenetrating

trauma may produce pneumothorax by bursting injuries to the lung, particularly when the force is applied to an expanded lung with the glottis closed. This would explain Dieffenbach's observation that most of the injuries of this type occur in children in whom the chest wall is more pliable.

The injuries in Cases 1 and 2 can well be explained on this hypothesis.

We have an additional case which we feel had the same mechanism of injury to lung. This case is not included with Cases 1 and 2 as there was evidence of incomplete fractures of several ribs. The fractures were of the green-stick type with only the inferior cortex being involved and could not adequately account for the severe injury to the lung. This case also demonstrates one of the more severe types of injury that can result from such a mechanism.

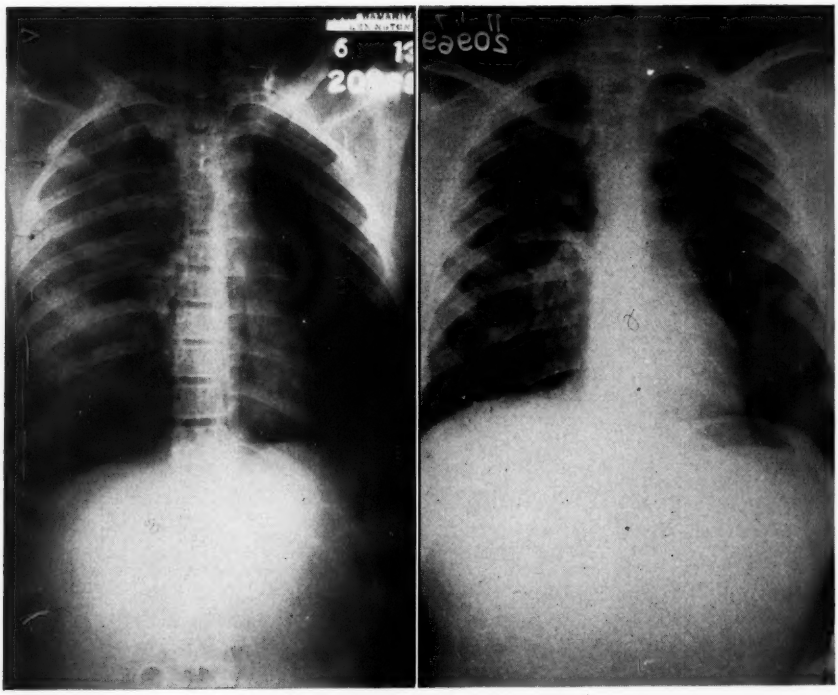

Fig. 3.—Roentgenograms show incomplete fracture of ribs and pneumothorax on right with re-expansion of lung after adequate therapy.

CASE 3.—K. T., a 14-year-old white boy, was admitted to the Good Samaritan Hospital on June 13, 1952, because he had fallen off a truck and it had run over his thorax. He was unconscious for 10 to 15 minutes. Except for occasional mild infections of the respiratory tract and the usual childhood diseases he had been in good health prior to the accident.

Physical examination on admission revealed a well-developed, well-nourished, acutely ill boy. There was a laceration of the right ear through the anterior margin across the canal and the entire ear was displaced posteriorly.

Examination of the chest showed the right side did not expand as well as the left. There were abrasions across the right anterior and posterior chest wall. The patient was

coughing up bloody sputum. The breath sounds were decreased over the right lung field. Systolic blood pressure was 100 mm. Hg, and diastolic pressure 70 mm. Hg. Pulse rate was 110 with sinus rhythm. The heart was not enlarged and no murmurs were audible. The remainder of the physical examination revealed no abnormalities.

Laboratory examinations revealed a red blood cell count of 5 million per cubic millimeter, hemoglobin 13.4 Gm. per 100 c.c., and leukocyte count 17,350 per cubic millimeter with 80 per cent segmenters, 14 per cent band forms, and 6 per cent lymphocytes. Urinalysis yielded a specific gravity of 1.025, no sugar or protein, and an occasional leukocyte on microscopic examination.

Roentgenograms of the chest exhibited incomplete fracture of the right fifth and sixth ribs with questionable fractures of the right second and third ribs. The superior cortex of these ribs appeared intact. There was an approximately 60 per cent collapse of the right lung as a result of pneumothorax (Fig. 3).

Under 1 per cent procaine infiltration anesthesia, a 22 French Foley catheter was inserted through the second intercostal space in the mid-clavicular line and attached to a water-seal drainage bottle.

The patient continued to pour out large quantities of air through the intercostal catheter. Twenty-four hours following insertion of the catheter he was found to have forced expirations and a respiratory rate of 50 per minute. The trachea was shifted to the left, and the left lung was wet in spite of the patient's effort to cough. His cough was productive of a small amount of bloody sputum.

On June 14, 1952, a right thoracotomy was performed through the right fifth intercostal space anteriorly. There was a moderate amount of blood in the pleural cavity. A large rent was found in the superior surface of the upper lobe which was approximately 12.5 cm. in length and 5 cm. in depth. The posterior segment of the lung was fairly firm and appeared to have a hematoma within the parenchyma of the lung. No active bleeding could be demonstrated. The wound of the lung was closed with interrupted silk sutures. The lung could then be expanded well except for the hemorrhagic portion of the upper lobe and there was no demonstrable air leak. No evidence of bleeding could be found. The pleural cavity was drained with two intercostal catheters, one anterior and one lateral. The pleural cavity was irrigated well with saline, and the wound was closed in layers with interrupted fine silk sutures.

The patient had an uneventful postoperative course and was discharged on the seventh hospital day. The lung remained expanded. The hematoma of the upper lobe gradually cleared over a period of about ten days, and since that time chest films have showed no abnormality of the lung.

SUMMARY

1. Three cases of traumatic pneumothorax are presented, two cases without evidence of rib fracture and one with incomplete fracture of ribs.

2. Three cases of traumatic pneumothorax have been presented which would substantiate the mechanism of injury suggested by Lindskog and Liebow.

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PERIPHERAL BRONCHIAL ADENOMA OF MUCOUS GLAND TYPE

Clinical and Pathologic Aspects

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Washington, D. C.

SINCE 1882, when H. Muller first described the tumor which has come to be known as bronchial adenoma, two major and opposed theories have been offered to explain its mode of origin. Churchill,[1] in 1937, noted a superficial resemblance of bronchial adenoma to fetal lung and suggested that bronchial adenomas may be vestigial lobes occurring in the bronchial wall. This concept was fully developed by Womack and Graham,[2] with the intriguing theory that most of the bronchial adenomas were mixed tumors and had an organoid multidermal origin from undeveloped anlagen of the lung. With the passage of time, however, the mixed-tumor theory has lost many advocates and the arguments against it have been convincingly summarized by Liebow[3] in these words: "The common appearance (of the tumors) is not that of alveolated tissue; cartilage appears to be engulfed or invaded rather than formed by the tumor and such bone as is found occurs either from metaplasia of pre-existing cartilage or within the septa or capsule of the tumor. It does not seem to be a part of the neoplastic process."

The second major theory, suggesting that bronchial adenoma arises from the bronchial mucous glands or their ducts, was proposed by Reisner[4] in 1928 and Kramer[5] in 1930. Both were of the opinion that the tumor cells more closely resembled the secretory ducts of the bronchial mucous glands than the alveoli and were probably derived from the former. Numerous investigators[6-13] have concluded that bronchial adenomas of the carcinoid and cylindroid types are derived from the bronchial mucous glands or their ducts. The great bulk of evidence offered in support of this theory has been *indirect* and is summarized as follows:

1. The anatomical location of the tumor in the wall of the bronchus and the presence of intact bronchial epithelium covering it strongly suggested origin from a structure normally found in the bronchial wall. The epithelial nature of bronchial adenoma would logically point to the bronchial mucous glands as the site of origin.

2. Bronchial adenomas apparently never arose from small bronchioles in which mucous glands were absent.

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3. Some investigators have seen "transitional cells" which were thought to bridge the gap between bronchial adenoma and the adjoining bronchial mucous glands. The neoplastic glands, however, did not closely resemble the bronchial mucous glands.

4. The presence of mucus in some of the tumors, either within the lumina of the neoplastic glands or within the cytoplasm of tumor cells, has lent some support for origin in the bronchial mucous glands. It should be noted, however, that the neoplastic glands containing mucus did not closely resemble the normal bronchial mucous glands.

Recently, the first bit of *direct* evidence in support of the theory of origin from bronchial mucous glands has been offered by Ramsey and Reimann,[14] who reported a bronchial adenoma which was clearly of mucous gland origin. In that tumor there was a striking resemblance of the neoplastic glands to normal bronchial mucous glands. We have recently observed a similar tumor. These two cases of bronchial adenomas clearly arising in and closely resembling bronchial mucous glands seem to provide a missing pathogenetic link in the theory of origin of bronchial adenomas from bronchial mucous glands.

CASE REPORT

J. H., a 66-year-old Negro farmer (retired), entered the District of Columbia General Hospital for the first time on Nov. 9, 1953, complaining of hematuria of eleven days' duration. History and physical examination on admission revealed signs and symptoms of prostatism of several years' duration. The patient underwent a retropubic prostatectomy on Nov. 17, 1953. The pathologic diagnosis was benign nodular hyperplasia.

Chest x-rays taken on Nov. 9, 1953, disclosed an unsuspected solitary nodular density in the right lung at the level of the third right anterior interspace (Fig. 1, *A* and *B*). This was interpreted as being due to either neoplasm or granuloma. On Jan. 18, 1954, the patient was readmitted to the District of Columbia General Hospital for resection of the solitary coin-shaped lesion (noncalcified) of the right lung.

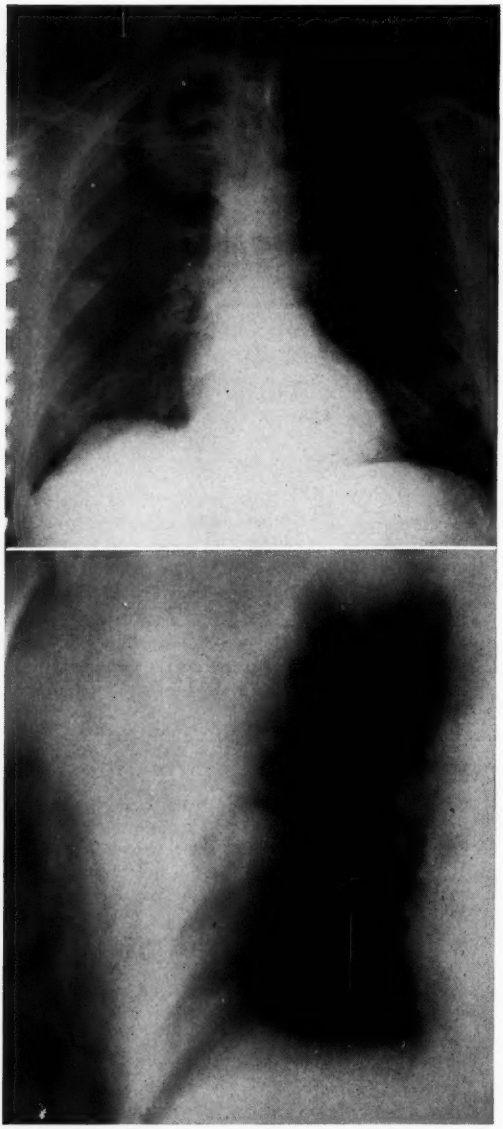
Additional history disclosed that the patient smoked one pack of cigarettes a day but denied any symptoms referable to the respiratory system. Tuberculin and histoplasmin skin tests were positive (1 plus).

On Jan. 21, 1954, the patient was operated on and the operative note follows.

Operative Note.—The skin incision was made on the right side along the fifth interspace. The pleural cavity was entered through the fifth interspace without removal of a rib. The rib spreaders afforded adequate exposure. Examination revealed a 2 cm. hard, firm nodule deeply embedded in the posterior part of the middle lobe. The tumor could not be seen but could only be palpated. There was no evidence of atelectasis. The posterior part of the middle lobe was freed from the upper and lower lobes to permit accurate approximation of two clamps to permit a wedge resection. The tumor mass was removed with a wide margin of safety. The severed edges of the lung were then sutured with two tiers of 000 chromic catgut on an Atraumatic needle. The first tier was a continuous mattress suture and the second tier was a whipstitch. No bleeding occurred and there was no leakage of air. Upon section of the tumor in the operating room it was believed to represent a metastatic tumor of undetermined origin. No further resection was deemed advisable. Positive pressure anesthesia was applied. The pleural cavity was drained by the water-seal method. The chest wall and parietes were closed in layers under positive pressure anesthesia.

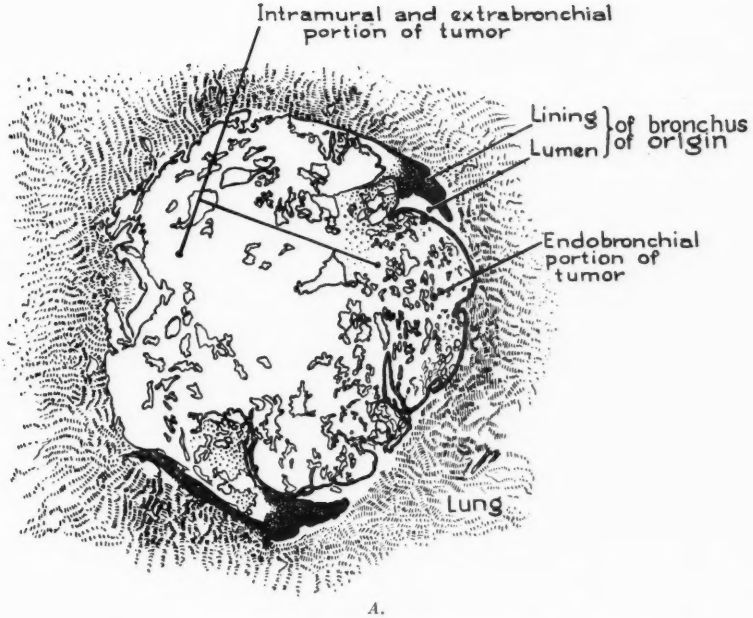
Because granuloma was suspected, a portion of the tumor removed surgically was sent to the bacteriologist for cultural studies. These were negative. The remainder of the specimen was submitted to the pathologist for gross and microscopic examination.

A.

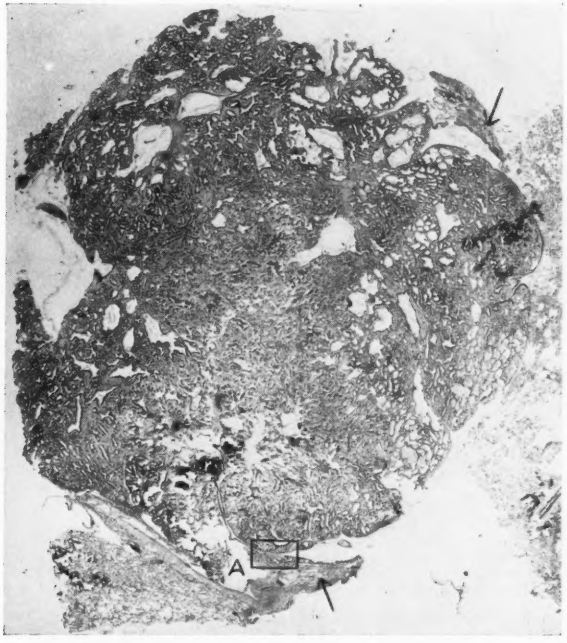


B.

Fig. 1.—Circumscribed solitary mass in the right mid-lung field, demonstrated by postero-anterior view (*A*), and tomogram (*B*).



A.



B.

Fig. 2.—*A*, Diagrammatic sketch of bronchial adenoma of mucous gland type. *B*, Low-power photomicrograph of tumor. Arrows indicate remnants of the lining of the bronchus of origin. Cystically dilated neoplastic glands are interspersed with smaller ones (×35; reduced 1/6.)

Fig. 3.

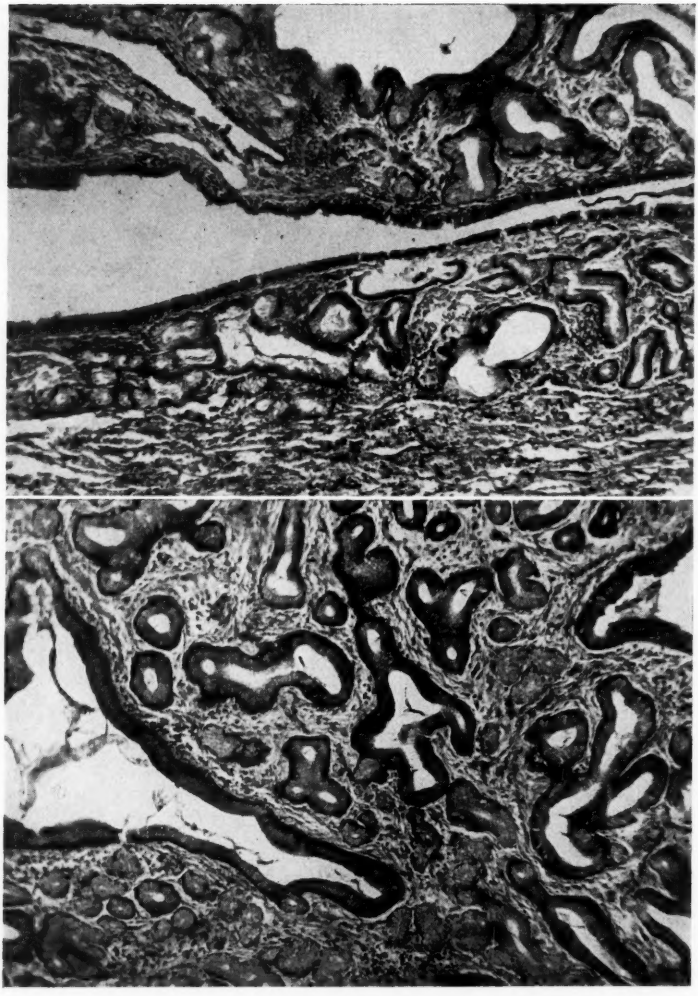


Fig. 4.

Fig. 3.—Enlarged photomicrograph of section *A* in Fig. 2, *B*. Below are hyperplastic bronchial mucous glands covered by bronchial epithelium. Above is a portion of the endobronchial part of the tumor also covered with bronchial epithelium. Note the close resemblance of the hyperplastic mucous glands to the neoplastic mucous-type glands above. (×150; reduced ⅓.)

Fig. 4.—Higher-power photomicrograph from a more central portion of the tumor. Note the larger cystically dilated mucous-type glands and the smaller adjoining glands. The nuclei are basal, flattened, and pyknotic, and the cytoplasm is foamy and rich in a mucicarmine-positive material. Mononuclear cells are present in the connective tissue stroma which surrounds the glands. (×160; reduced ⅓.)

Gross Pathology.—The specimen consisted of a wedge-shaped portion of right middle lobe taken from the periphery. It measured 4 by 3 by 1 cm. Five-tenths centimeter beneath the pleural surface there was a tan, slightly trabeculated, spherical tumor which measured 2.0 cm. in diameter. The cut surface was slightly moist and mucoid. The tumor appeared to be well demarcated from the surrounding lung parenchyma although no true capsule was seen. There was no apparent relationship of the tumor to adjoining bronchi. A frozen section was done because clinically there was a suspicion of metastatic carcinoma, possibly from the previously resected prostate gland. The frozen-section diagnosis was "metastatic carcinoma, primary site undetermined." The paraffin-section diagnosis was "bronchial adenoma of mucous gland type."

Microscopic Examination.—Approximately one-third of the tumor lay within a dilated bronchus, while the remaining two-thirds was intramural and extrabronchial (Fig. 2, *A* and *B*). The endobronchial portion was polypoid and covered by pseudostratified ciliated columnar epithelium which was mostly intact. Beneath the lining epithelium of the bronchus and lying in its wall were hyperplastic, occasionally dilated mucous glands which were morphologically indistinguishable from the mucous glands comprising the tumor (Fig. 3). The tumor was composed almost entirely of mucous-type glands between which was a fine areolar connective tissue fairly rich in mononuclear cells and containing lesser numbers of plasma cells and lymphocytes (Fig. 4). The mucous glands varied in shape from round and oval to tortuous. They all possessed lumina which contained varying amounts of a stringy mucoid material staining pink with mucicarmine. The lining of the glands was usually one cell in thickness, tall columnar epithelium, and the cells had basal nuclei and abundant foamy cytoplasm which stained intensely red with mucicarmine. The nuclei which rested on the basement membrane were small, pyknotic, flattened or ovoid, and closely resembled the nuclei of the bronchial mucous glands. Mitoses were not observed. No ciliated cells were seen. Thin-walled blood vessels were present but not in abundance. Within the intramural portion of the tumor was a fragment of degenerating hyaline cartilage, probably a remnant of the cartilage of the bronchus of origin. The tumor was separated from the surrounding lung parenchyma by an ill-defined capsule composed of condensed connective tissue fibers. The small bronchi and bronchioles adjoining the tumor appeared somewhat dilated. A portion of lung parenchyma adjoining the tumor contained many alveoli which were filled with stringy mucoid material resembling the mucoid material seen within the lumina of the neoplastic mucous glands.

CLINICOPATHOLOGIC ASPECTS

We were able to find only one other case in the literature of bronchial adenoma which was pathologically of the mucous gland type and identical with

TABLE I. CLINICAL AND PATHOLOGIC ASPECTS OF THE PRESENT CASE COMPARED WITH THE PREVIOUS REPORTED CASE OF RAMSEY AND REIMANN

CASE OF RAMSEY AND REIMANN	PRESENT CASE
Clinical Comparison	
X-ray: atelectasis	X-ray: coin lesion
Bronchoscopy: visualized	No bronchoscopy
Signs and symptoms of infection (fever, general aching, etc.)	Asymptomatic
54 years old	66 years old
Race: White	Race: Negro
Pathologic Comparison	
Tumor arose in main bronchus, right lower lobe	Tumor arose in small peripheral bronchus, right middle lobe
Tumor was entirely endobronchial	One-third of tumor was endobronchial, two-thirds intramural and extrabronchial
Tumor was 1.3 cm. in diameter and pedunculated	Tumor was 2 cm. in diameter and non-pedunculated
Bronchial adenoma, mucous gland type	Bronchial adenoma, mucous gland type
Neoplastic glands were mucicarmine-stain positive (presumptive evidence of mucus)	Neoplastic glands were mucicarmine-stain positive (presumptive evidence of mucus)

our case. This is the case reported by Ramsey and Reimann in 1953. A comparison of the clinical and pathologic aspects of the two cases is summarized in Table I.

The clinical differences may be ascribed to the difference in location of the two tumors. The case of Ramsey and Reimann had clinical features which have been described in cases of adenomas located in large bronchi, while the clinical aspects of our case closely resembled those described in cases of bronchial adenomas arising in small bronchi. The clinical and pathologic description of bronchial adenomas arising in small bronchi is a relatively recent event and as late as 1948 Willis[15] stated, "Bronchial adenoma is almost invariably situated in a large bronchus close to the hilum, never in the periphery of the lung."

Maier and Fischer[16] have reported five cases of bronchial adenoma arising in small bronchi and three were clinically asymptomatic. Good and Harrington[17] reported 23 additional cases arising in small bronchi, 17 of which were asymptomatic. All were of the carcinoid type. These authors were of the opinion that bronchial adenomas arising in small bronchi close to the hilum of the lung were more likely to produce symptoms than adenomas located at the periphery.

The roentgenographic appearance of the tumor in our case (peripheral bronchial adenoma of mucous gland type) closely resembled the roentgenographic appearance of bronchial adenomas arising in small bronchi as reported by Maier and Fischer and by Good and Harrington. The x-ray appearance was that of a "solitary intrapulmonary tumor." This designation is used to represent any lesion whether neoplastic, inflammatory, granulomatous, developmental, or vascular that is located within the lung and is round, oval, or slightly lobulated, with a smooth contour sharply demarcated from the normal lung. Our studies on approximately 200 cases have indicated the futility of attempting to establish a precise preoperative diagnosis of a circumscribed noncalcified intrapulmonary lesion. For this reason, the early use of exploratory thoracotomy as a diagnostic measure is used in all cases of noncalcified circumscribed masses. In a series of 156 cases of solitary mass lesions of the lung reported by Good, Hood, and McDonald,[18] 12 (8 per cent) were bronchial adenomas.

CLASSIFICATION

The currently accepted classification of bronchial adenomas into carcinoid and cylindroid groups does not include bronchial adenomas of mucous gland type. The carcinoid tumors were so named because of their resemblance to carcinoid tumors of the gastrointestinal tract, while the cylindromas derived their name from a group of benign salivary gland tumors which they closely resembled. This classification has been modified by Liebow to include an oncocytoid variant of carcinoid tumors and a mucoepidermoid variant of cylindromatous tumors. Moreover, the literature on bronchial adenoma includes other "exceptions" to the current classification. For example, reference is made to a small round cell variant of cylindroma[19] which resembles "oat cell carcinoma." Two other variants of cylindroma-basal cell carcinoma and epithelioma adenoides cysticum have been described by Holley.[20]

It seems that all bronchial adenomas are channeled into either the carcinoid or cylindromatous groups regardless of how well they fit. It is questionable whether our knowledge of bronchial adenoma is abetted by lumping several pathologically heterogeneous tumors into the carcinoid and cylindromatous groups. We believe that an improved pathologic classification of bronchial adenoma based upon a study of a large number of cases would be valuable.

MALIGNANCY OF BRONCHIAL ADENOMAS

The tumor in our case was unusually well differentiated into mucous type glands and appeared to be entirely benign. No regional lymph nodes were available for histologic study and no nodules were felt in the lungs at the time of operation. Ramsey and Reimann believed that their tumor was also entirely benign. Opinions regarding the degree of malignancy of bronchial adenomas of the carcinoid and cylindromatous types run to extremes. For example, Womack and Graham regarded many of them as potentially malignant and believed that some of the bronchogenic carcinomas arose in pre-existing adenomas by malignant transformation. Wessler and Rabin[21] and Rabin and Neuhof,[22] on the other hand, regarded most bronchial adenomas as benign lesions.

According to Moersch and McDonald, carcinoid tumors comprise 90 per cent of bronchial adenomas and cylindromas comprise 10 per cent. A difference in the relative malignancy of these two groups has been postulated by McDonald[23] on the basis of a greater infiltrative tendency on the part of the cylindromas, and McBurney, Kirklin, and Woolner[24] found that cylindromas metastasized three times as often as carcinoid tumors. Liebow, however, has pointed out that only a very few instances of *distant metastases* of the cylindroid variety have been reported, and he indicated that more data are needed before the question of the relative malignancy of the carcinoid and cylindroid types can be decided.

Recently McBurney, Kirklin, and Woolner reviewed all reported cases of "metastasizing bronchial adenoma." They were able to find 87 cases, of which 46 showed regional node metastasis only. The remaining cases had metastases in order of frequency to the liver, opposite lung, ipsilateral lung, pleura, esophagus and other mediastinal structures, bone and miscellaneous organs including brain, kidney, and adrenal gland. The incidence of metastasis of all reported cases was 10 per cent. The authors did not include data with regard to duration of life following discovery of the tumors and post-mortem findings with regard to the extent of tumor dissemination and the cause of death. We believe that these data are important in evaluating the degree of malignancy of bronchial adenomas. The authors contributed nine cases to the study and none of these included pathologic material or data from post-mortem examination.

The definition of malignancy in cases of bronchial adenoma has been a source of great confusion. Many pathologists and clinicians have considered metastasis to the regional lymph nodes as positive evidence of malignancy even though most cases of this type have had as benign a clinical course as cases without regional lymph node metastases. We believe that "malignant bronchial adenoma" should be defined as an adenoma which will metastasize distantly, and eventually cause death by metastasis.

The recognition of bronchial adenomas which metastasize distantly and cause death by metastasis is beset with many difficulties. Many of the published reports of "metastasizing bronchial adenoma" are deficient in one or more of the following data: (1) post-mortem findings to establish the metastasizing bronchial adenoma as the cause of death, and to rule out (a) a bronchogenic carcinoma arising elsewhere in the lung and (b) an extra-pulmonary malignancy undetected during life; (2) photomicrographs histologically comparing the original biopsy with distant metastases; (3) review of the pathologic material by one or more independent pathologists to establish concurrence in the interpretation of the biopsy or autopsy material; (4) duration of life following the discovery of the tumor.

In conclusion, we believe that many of the cases published as examples of malignant bronchial adenoma are incompletely documented and, therefore, the exact incidence of malignant bronchial adenoma remains undetermined. Carlens and his associates[25] in a recent report of 70 cases of bronchial adenoma have come to similar conclusions.

TREATMENT

No definite conclusions regarding the treatment of bronchial adenomas of mucous gland type are possible on the basis of two cases. Our case was treated by wedge resection because of the peripheral location of the tumor and Ramsey and Reimann's case was treated by segmental resection of the right lower lobe because of the central location of the tumor.

We believe that the treatment of choice in cases of bronchial adenomas of mucous gland type is conservative surgery since the tumors appear to be entirely benign histologically. The type of operative procedure chosen would depend upon the location of the tumor rather than upon the pathologic subtype. Peripheral tumors may be removed by wedge resection, while central tumors may be removed by segmental resection, partial or total lobectomy.

SUMMARY AND CONCLUSIONS

1. A case of bronchial adenoma clearly arising in and closely resembling bronchial mucous glands is reported. The tumor was located at the periphery of the right middle lobe and arose in a small bronchus.

2. The histologic appearance of this tumor lends *direct* support to the theory that bronchial adenomas of the carcinoid and cylindroid types arise in bronchial mucous glands. Most of the evidence previously advanced to support this theory has been indirect.

3. Bronchial adenomas arising in small bronchi are usually asymptomatic and are more readily detected by x-ray than by bronchoscopy.

4. Bronchial adenomas of mucous gland type do not readily fit into the current classification of bronchial adenomas, and other exceptions are noted. There exists a need for reclassification of bronchial adenomas.

5. Bronchial adenomas of mucous gland type appear to be entirely benign. The exact incidence of "malignant" bronchial adenomas of the carcinoid and cylindroid types is undetermined. Many cases so reported are incompletely documented.

6. Treatment of choice in bronchial adenomas of mucous gland type appears to be wedge resection, segmental resection, or total lobectomy, depending upon the location of the tumor with respect to the hilum of the lung.

We wish to thank Dr. Samuel H. Rosen, Chief of Section of Pulmonary and Mediastinal Pathology, Armed Forces Institute of Pathology, Washington, D. C., for his many helpful suggestions and criticisms.

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THE SURGICAL MANAGEMENT OF FUNNEL CHEST

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SINCE the comprehensive review of the literature on funnel chest by Ochsner and DeBakey[1] in 1939, the attention of many surgeons has been directed to the problems of etiology and therapy of this deformity. Brown in 1939 first championed the cause of the hypertrophied substernal ligament as an etiological agent, although such a structural defect had been noted by Spitzy,[2] Hofbauer,[3] and Hutchinson[4] many years previously. It was Brown[5] who first described the minor ligament cutting operation which could be employed successfully in infants, as well as a more extensive cartilage severing operation in the larger defects of older children and adults. The papers of Lester[6-8] have very clearly outlined details of the surgical corrective technique which in our hands has proved very satisfactory and effective, and which we have employed with a few minor modifications over the past seven years.

Various other papers[9-11] have reported the experiences of other surgeons with small series of cases employing essentially similar operative techniques with the usual minor modifications. It appears that there is rather universal agreement that the deformity is not as uncommon as once thought; that there is an hereditary factor in the etiology, although some cases occur without familial history of the deformity; that the deformity is usually present from birth, although it does not progress in some instances; and that small deformities can be corrected by a simple operation at an early age. It has been shown[12-14] that there is definite impairment of pulmonary function in some instances which can be corrected by surgery, and that cardiocirculatory impairment can be produced by extreme degrees of deformity, especially in the older age group. These, too, can be improved by correcting the sternal aberration.

It is obvious, therefore, that further reports of small series of successfully treated cases can be of no great value to students of the funnel chest problem because it has now been adequately demonstrated that there is need for the corrective surgery, and that it can be performed universally with success and with negligible mortality.

It has been our conviction from the time of the early reports that the most confusing aspect of the entire problem has been that of the proper selection of cases and the optimal age for operation. In other words, which cases should we do, and when shall we do them? That problem can be resolved only by observation of large series of cases, of those operated upon and those not operated upon, observed over a period of years.

It has been a universal experience, I am sure, for surgeons all over the country to be confronted by a variety of infants with a variety of degrees of

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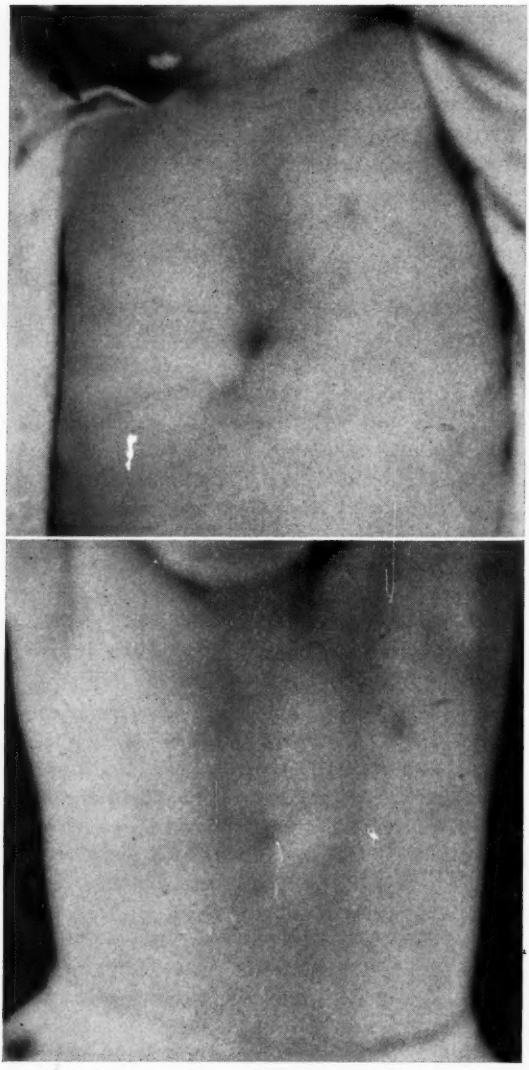
thoracic deformity ranging from dimples to deep depressions. One does not ordinarily advise surgery in a very young infant with a very small defect and is rewarded by the disappearance of the dimple in about one-half of these patients and chagrined by the steady growth, deepening and widening of the defect in others. The question then is: Does one do 50 per cent unnecessarily or deny surgery unnecessarily in 50 per cent only to regret having procrastinated and thus having lost the opportunity to perform a simple operation instead of the large operation entailing resection of a greater number of costal cartilages? We have not been able easily to foretell at the age of one month which small defect was going to enlarge. In the case of the obviously larger defects, there is no problem. As Brown[15] has stated, these cannot be done early enough, and occasionally the small operation with resection of no cartilage will suffice. Unfortunately we have encountered only two ideal cases for the small operation. In the others, the deformity was either too small to warrant surgery, or so large that the more extensive operation was mandatory even at a very early age. It has been our experience that infants do not tolerate the extensive resection of costal cartilages necessary in the performance of the large operation, even with adequate fixation of chest wall with anterior struts. It has been seen from our series of cases that deformities classified as moderate to large in size during the first months of life do not ordinarily increase in size appreciably by the first year, and delay of surgery until the infant has reached the age of 12 months does not ordinarily necessitate a more extensive operation than would originally have been necessary. The resiliency of the costal cartilages prior to the age of 5 years allows the chest wall to assume rather normal contours once the sternum is elevated. We have not encountered fixed sternal deformities prior to the age of 18 months. If the deformity is markedly symptomatic, or increasing rapidly in size, surgery is promptly performed.

It has been our observation that the infants under 12 months of age with small defects can be classified roughly into those without upper respiratory obstruction and those with obstruction in the form of congenital hypoplasia of the larynx, congenital stridor, hypertrophy of lymphoid tissue (tonsils, adenoids, etc.), or other causes. It is absolutely mandatory, as Lester[7] stated, that such obstruction be relieved before correction of the funnel chest is attempted. If repair of the funnel is done in the presence of respiratory obstruction, the likelihood of recurrence of the deformity is great. In a fair proportion of those children with small deformities, there will be some diminution in size of the deformity once the obstruction is relieved. In at least two of our nonoperative cases there has been sufficient diminution in size to warrant abandonment of the decision to perform surgery.

On the other hand, our very first case demonstrated that restoration of the normal mechanism of respiration by repair of a funnel deformity was necessary before a 6-week-old infant with laryngeal stridor could breathe sufficiently well to be removed from an oxygen hood. Paradoxical motion of the thorax was marked. There was (Fig. 1, *A* and *B*) contraction of the lower thorax and both costal arches as well as retraction of the sternum on inspiration prior to operation. Immediately after resection of the seventh and eighth costal car-

A.



B.

Fig. 1.—*A*, Photograph of the funnel chest deformity of a six-week-old infant who was unable to maintain oxygenation out of an oxygen hood prior to operation. *B*, Photograph of child shown in Fig. 1, *A* two months postoperatively.

tilages bilaterally with section of a hypertrophied substernal ligament, the paradoxical motion ceased, and with hyperextension of the neck, the child was able to breathe normally. In this instance, it was felt that the operation was lifesaving in that it provided adequate respiratory exchange for the first time since birth. We have had no other cases in which the changes created by operation were so dramatic, although the paradoxical movement of the lower thorax is markedly diminished in all cases immediately following correction of the deformity. In this regard, it may be well to mention that stripping the anterior diaphragmatic insertions downward from the periochondrial beds of the resected cartilages of the costal arch seems a most important part of the operation and is the most effective maneuver in the reduction of diaphragmatic tug on the costal arch and lower thorax, thereby greatly lessening paradoxical motion. Even though paradox did not disappear immediately in all instances, it was no longer present by the time the child was discharged from the hospital—a period usually not exceeding two weeks.

The presence, absence, or degree of paradoxical motion of the lower thorax has been one of the influencing factors in reaching a decision whether to recommend surgery or not in the younger infants. In those infants having small defects, with little or no paradox, it has been our policy to withhold surgery and to observe the infant at monthly intervals, carefully measuring the size of the defect. In over 50 per cent of these infants the defect disappeared. It was found that without exception the greater the degree of paradoxical motion, the more likely the defect was to persist or to enlarge. This observation corroborates Sweet's[16] impression on reporting his first two cases. In those cases in which the defect was found to remain stationary, operation was postponed until after the first year of life. In those cases in which there was appreciable increase in the size of the deformity, immediate surgery was done. We have been so impressed by the relationship of paradox to the continued growth of the defect, that we have recently come to urge surgery upon every infant with small defects who demonstrated marked contraction of the costal arches on inspiration. This attitude has resulted from the fact that in no instance in which there was marked paradox did the defect remain stationary. All of such defects continued to enlarge over the period of observation. This factor will undoubtedly result in a larger percentage of early operations in our series in the future. (There have been only five under 6 months to date, as contrasted to ten in the 6- to 12-month age group.)

After the date on which Lester[6] stated that paradox is present in these patients even when an endotracheal tube is in place, we have routinely taken a moment or two before beginning operation to note that this is universally true, although paradox is not quite as marked in the anesthetized intubated patient as it is when the child is awake and sitting erect.

We have not encountered fixed deformities of the sternum in the age group under 2 years, other than the sharp angulation of the xiphoid process. This process has been resected of necessity in almost all instances. After this age, fixed deformity of the sternum of some degree is the rule, and must be dealt with. It is almost needless to repeat the observation regarding posture which

has been made many times before, namely: the round-shouldered, potbellied profile which accompanies the sternal deformity which becomes noticeable as soon as the child assumes the upright habitus and which becomes more strikingly apparent with each passing month.

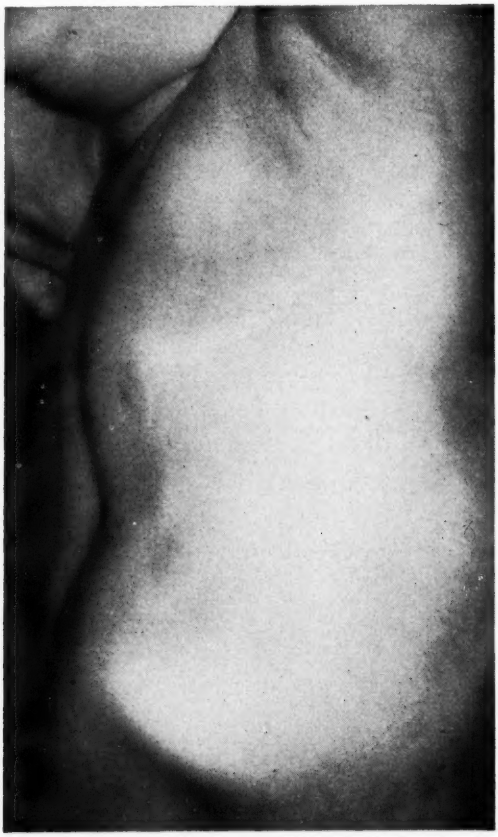


Fig. 2.—Photograph of a child having a marked transverse groove deformity accompanying a funnel depression of the sternum.

In some instances there are, in addition to the sternal depression, transverse grooves and ridges of the thorax which must be dealt with by the resection of longer sections of costal cartilages and ribs (Fig. 2).

The purpose of this paper is to report our observations on 51 operative cases, and ten cases upon whom operation was judged unnecessary at first examination.

Risk of Operation.—There were no operative deaths. Only one complication, in the form of a minor wound infection, was encountered in the series.

The age incidence of surgical cases is tabulated in Table I. It is seen that the largest number of surgical procedures were done in the age group between

6 months and 2 years. As has been mentioned, it is our opinion that the ideal age for surgery is between the twelfth and twenty-fourth months. Probably the bulk of operations done in the future will be done upon this age group. At this age the child appears to be able to better tolerate anesthesia, as well as the paradoxical respiration that occurs following removal of costal cartilages and sternotomy. The fixed deformities of sternum have not yet occurred at this early age, and so waiting until the child reaches this age does not necessitate a more extensive procedure.

TABLE I. AGE INCIDENCE

AGE	NUMBER
Under 6 months	5
6-12 months	10
1-2 years	13
2-3 years	8
3-4 years	5
4-5 years	0
Over 5 years	10
Total	51

TYPE OF OPERATION

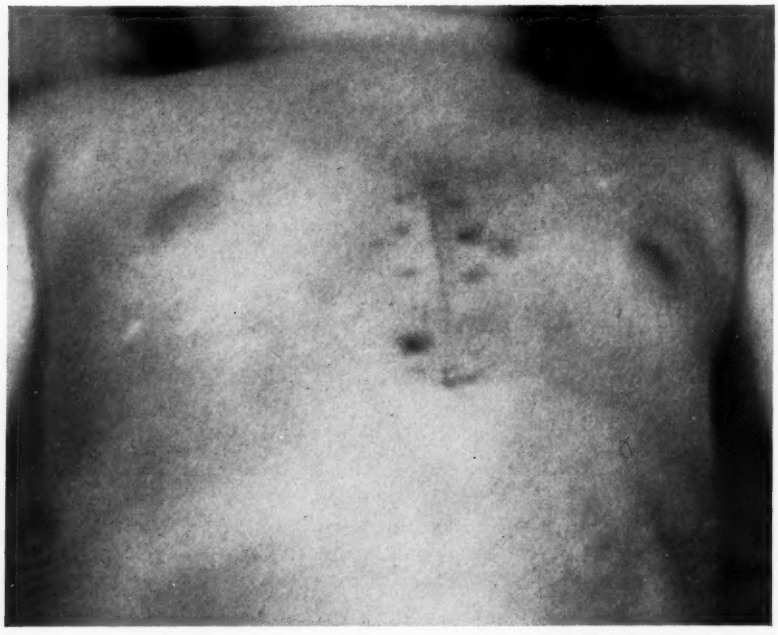
The simple ligament-cutting operation was done in only two instances, both in children not over 4 months of age. One of these patients was described above —our first patient in the series, in whom paradoxical motion of the thorax was so marked that prior to operation the child could not be removed from the oxygen tent. Following the simple operation, respiratory movement became immediately more effective, and the child has undergone remarkable growth and development (Fig. 1, *B*).

The only other simple operation was performed upon a 4-month-old infant with a small defect. The operation upon this patient was done early in the series, and probably at the present time the patient would be observed until the age of 1 year. In this instance a portion of the deformed costal arches was removed bilaterally. The child has done well.

In forty-nine other cases the extensive subperichondrial resection of costal cartilages has been done. All of the deformed cartilage—sometimes this involves resection of the entire cartilaginous portion—of the rib was removed. The number of cartilages removed depended upon the number involved in the deformity and varied from segments of three to six or seven cartilages bilaterally. We have been particularly careful to remove all of the involved portion of each cartilage, preferring to remove an excess rather than an insufficient amount since regeneration promptly takes place if subperichondrial resection is done, and stability of the chest wall returns in a matter of ten days in almost all instances. This procedure, we feel, has been an important factor in eliminating the necessity for second operations for recurrent deformity. There have been no recurrences in the entire series of cases. All cases are being observed at least yearly. The longest period during which any case has been under observation is seven years.

We believe that it is extremely important to perform subperichondrial resection for two reasons: first, regeneration of cartilage or bone occurs rapidly,

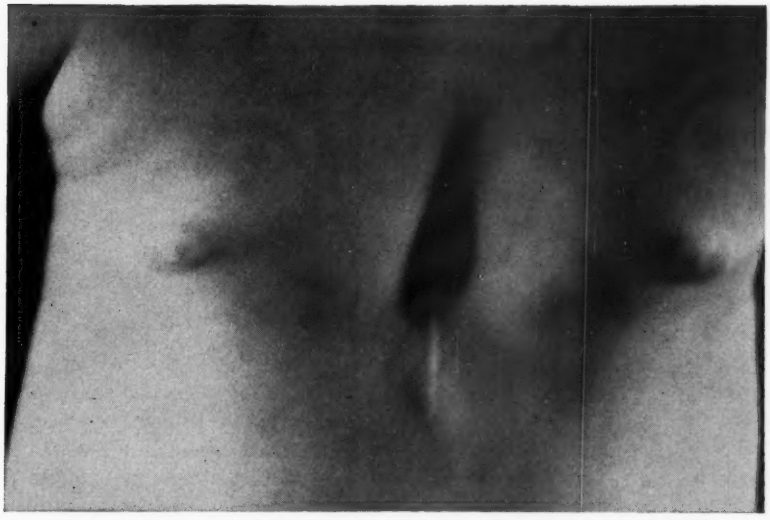
and adequate stability of the chest wall is assured in less than two weeks; second, by judiciously placing sutures in the perichondrium of resected ribs the shape of the chest wall can be altered within limits so that external fixation of the sternum is scarcely necessary to ensure regeneration of cartilage in the corrected position. In other words, the shape given to the anterior chest wall by carefully placed sutures in the perichondrium was made permanent by cartilaginous regeneration.



A.

Fig. 3.—*A*, Photograph of the chest of a four-year-old child, two weeks after operation showing adequate correction of the deformity. *B*, Photograph of same child as in Fig. 3, *A*, two years postoperatively showing apparent recurrence of the funnel deformity due solely to poor posture. *C*, Photograph of the same child as Fig. 3, *A* and *B*, two months after physical therapy in the form of posture training, breathing, and shoulder exercises, showing disappearance of the deformity apparent in Fig. 3, *B*.

Another factor which we have felt to be of great importance in avoidance of recurrence of the funnel deformity is postoperative physical therapy in the form of breathing exercises, posture training, and shoulder exercises. Of course, all of these measures cannot be instituted in the very young age group because of lack of cooperation, but shoulder exercises and posture training can be accomplished even in the 2-year-old. The potbellied, round-shouldered habitus of the child with funnel chest has been described many times, and it has been our experience that the round shoulders and potbelly often remain a long time after the funnel has been corrected. Fig. 3, *A-C* shows a child, 2 weeks postoperatively (*A*), two years after resection (*B*), and several months after posture training had been reinstituted (*C*). In this instance the referring pediatrician had re-



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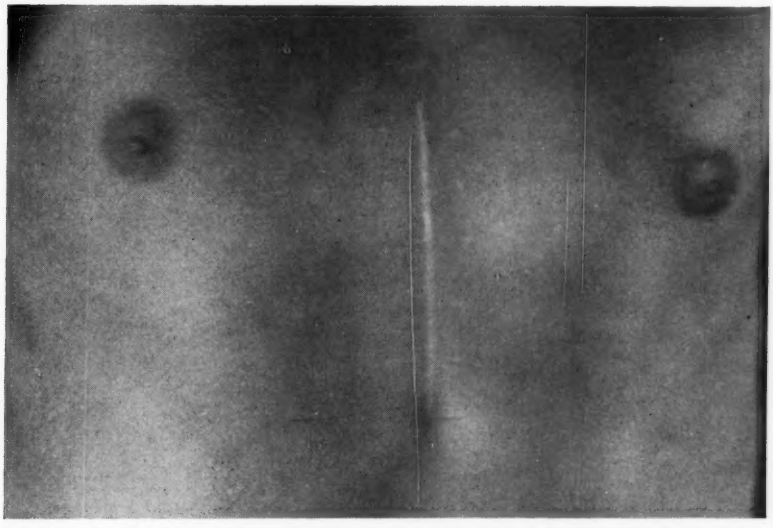


C.

Fig. 3, *B* and *C*. (*See legend on opposite page.*)

turned the patient to us for consideration of a second operation. As can be seen from the photographs simple exercises were the only measures necessary to restore the normal appearance of the chest. There was, in this instance, no deformity of the regenerated cartilages. Physical therapy will not, of course, correct deformed cartilages and is of no value when such deformities exist.

ANESTHESIA

Intratracheal light plane anesthesia with generous amounts of oxygen was employed in all cases as has been our practice in all thoracic surgery at Milwaukee Children's Hospital. We have encountered no laryngeal edema attributable to the tube in any case and have felt extra security in the knowledge that the anesthesiologist was adequately prepared for the inadvertent opening of the thorax which occasionally takes place in these operations. Although positive pressure can be given with a tight-fitting face mask, we have always felt that an intratracheal tube was much more adequate.

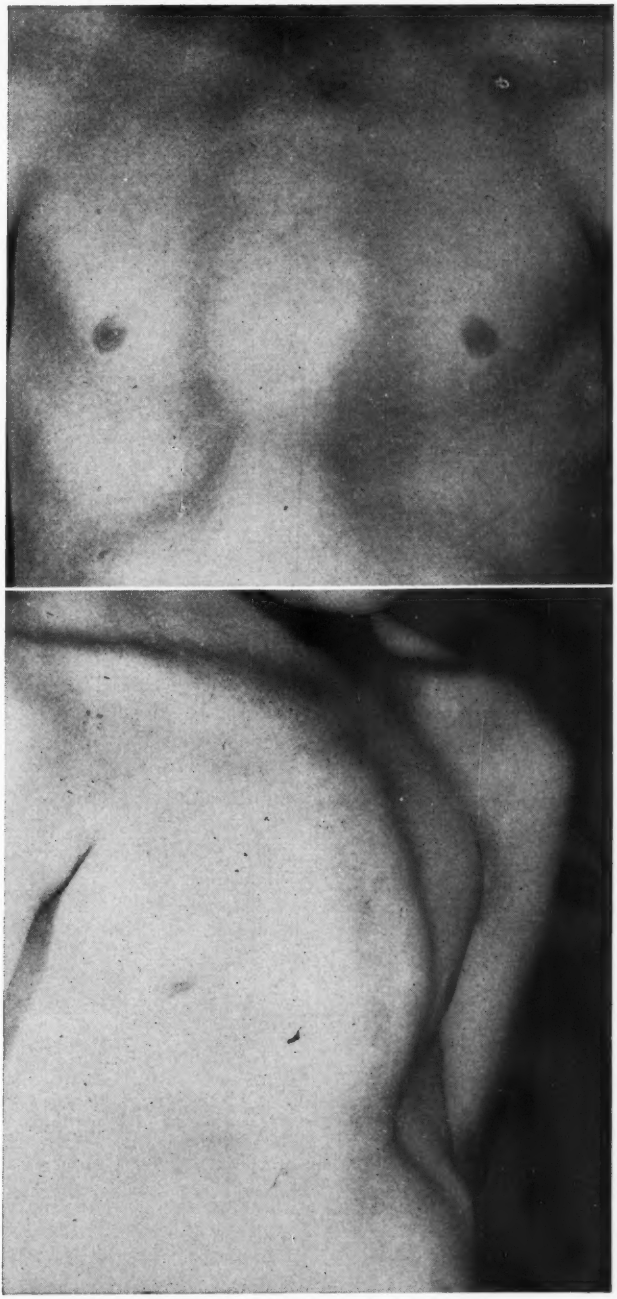
AGE AT OPERATION

It can be seen from Table I that over 50 per cent of our patients were operated upon prior to the age of 2 years. Operations upon the older age groups do not represent postponed surgery in any case, but were done later in life because the patient was seen for the first time at the more advanced age. The oldest patient in the series was 29 years old; operation was done because of impaired respiratory reserve. The result was satisfactory but not spectacular, and there is some question as to the improvement in respiratory function. Maximum breathing capacity increased from 75 to 95 liters per minute (scarcely a spectacular improvement, but the patient is subjectively improved).

SUBSTERNAL LIGAMENT

Hypertrophy of the substernal ligament of significant degree was noted in only thirteen cases in this series. Thickening of sufficient magnitude to cause abnormal traction on the lower sternum was observed in only five cases. A sufficient amount of thickening to warrant the term hypertrophy was noted in eight other cases. In thirty-eight cases there was no appreciable thickening or change in the ligament which differed from the structure observed in autopsy specimens of similar age. It is difficult, therefore, to attribute the deformity to the pull of this ligament on the sternum. It has seemed to us that as Sweet[16] has suggested, "the deformity is primarily skeletal." It appears that there is actually an overgrowth of costal cartilage which in its growth has forced the sternum backward toward the spine in the case of funnel chest, and which in the allied condition of pigeon breast has forced the sternum anteriorly (Fig. 4, *A* and *B*). Following resection of the deformed cartilaginous segments, the maneuver of elevating the sternum to its normal position reveals that the lateral sternal borders are almost, if not actually, in contact with the ends of the unresected cartilaginous or osseus rib (Fig. 5, *A* and *B*). This observation would tend to give weight to the concept that the deformity is caused by an overgrowth of cartilage rather than by ligamentous or muscle "pull." There is almost a superfluity of length of periosteum which has covered the deformed resected cartilage. This is usually reefed or "gathered" in the course of the repair, thereby elevating the costal arch and suspending the anterior chest wall from the upper resected ribs in such a way that the wall is quite stable even before regeneration takes place. When regeneration does progress, it proceeds from the perichondrium and periosteum of the resected ribs now lying in the cor-

A.



B.

Fig. 4.—*A* and *B*, Photograph of a nine-year-old boy, showing pigeon breast type of deformity due to overgrowth of costal cartilages and outward bowing of the cartilages with no deformity of the sternum itself.

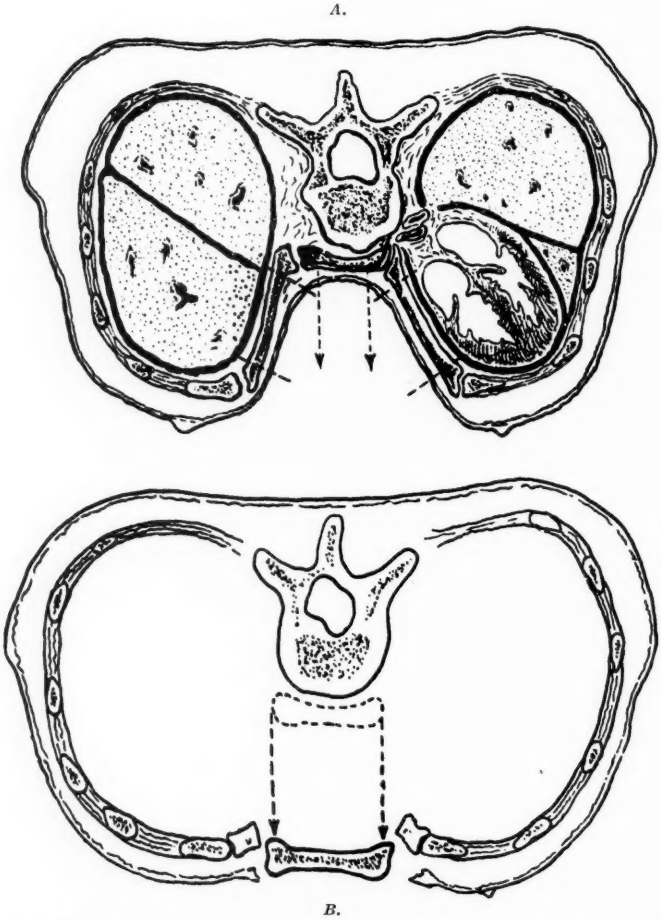


Fig. 5.—*A*, Diagram of a cross section of the thorax showing deep depression of the sternum and indicating between the broken line the amount of costal cartilage to be resected. The arrows indicate the distance which the sternum will be moved anteriorly following transverse osteotomy. *B*, Diagram of a cross section of the thorax showing the position of the sternum following resection of deformed costal cartilages bilaterally, transverse sternal osteotomy and elevation of the sternum to the corrected position. Note that the lateral sternal margins are almost in direct contact with the short remnants of costal cartilages and, in some cases where the entire length of the costal cartilage has been resected, are in contact with osseus rib.

rected position. Although no secondary operations were performed which would allow direct inspection of the regenerated cartilages, examination of these cartilages by palpation would indicate that the regenerated segments are somewhat tortuous as a result of the reefing, but that the contour of the chest wall approaches the normal. It is felt that the tortuosity of the regenerated segments adds stability to the chest wall.

AFTERCARE

It is not sufficient merely to correct the costochondral and sternal defects by resection. In those children who have assumed the round-shouldered, pot-bellied posture it is important to institute postural training, breathing and shoulder exercises which are calculated to encourage throwing back of the shoulders, elevation of the thorax and diaphragmatic rather than abdominal breathing. These exercises are begun on the third or fourth day following surgery and are continued for variable periods until such time that the child overcomes his tendency to slouch with consequent dorsal kyphosis and protuberant abdomen.

A course of physical therapy is given in each case until discharge of the child from the hospital, and his exercises are continued at home under the direction of the mother until the child has acquired the habit of maintaining proper posture.

UNOPERATED CASES

During a period of seven years, ten children were seen on whom surgical correction was not deemed advisable (Table II). Two of these had congenital laryngeal stridor. It was felt that the deformity of the chest was secondary to the stridor. In both of these patients seen at the age of 1 month and 17 months, respectively, the deformity subsequently decreased in size to nonsurgical proportions following relief of the stridor.

TABLE II. UNOPERATED CASES

AGE AT FIRST CONSULTATION	REASON FOR DELAY OF OPERATION	PRESENT STATUS
1. 1 month	Laryngeal stridor	Minimal deformity
2. 17 months	Laryngeal stridor	
3. 1 month	Defect too large for simple operation Too young for large operation	Minimal deformity
4. 1 month		Minimal deformity
5. 1 year		Minimal deformity
6. 2 years	Deformity too small to warrant surgery	Minimal deformity
7. 5 years		Minimal deformity
8. 2 months	Exstrophy of bladder	Deformity still present
9. 9 months	"Celiac disease"	Deformity still present
10. 2 years	Tetralogy of Fallot	Deformity still present

In the case of two infants seen at the age of 1 month with moderately large defects, on whom the larger operation seemed mandatory, surgery was to be postponed until they had reached the age of 1 year (the age which we considered ideal for the larger operation). Surprisingly, on the follow-up examination at the age of 1 year, the defects had decreased to such an extent that it was felt

surgery had better be postponed until further observation had established whether the improvement were going to continue. Both are now over 2 years old and appear to have only minimal deformities.

Three children, 1 year, 2 years, and 5 years of age, respectively, with very small defects were observed over a period of several years. The deformity did not increase in size in any of these, and although surgery was offered for purely cosmetic reasons, it was refused by the parents.

Three infants who had additional severe congenital defects (tetralogy of Fallot, exstrophy of the bladder, celiac disease) are still being observed, and corrective measures will probably be taken when and if the other problems are resolved.

SUMMARY

Observations gained from the study of fifty-one operated and ten non-operated cases of funnel chest are presented. This group of cases substantiates the fact that morbidity and mortality of surgical correction are negligible. The corrective surgical technique is fairly well standardized and productive of very satisfactory results in most instances.

The criteria for proper selection of cases for operation with particular reference to the ideal age and the type of operation indicated are discussed. The etiological role of the substernal ligament is discussed. In the light of our inability to demonstrate hypertrophy of this structure in most instances, we have been led to conclude that the deformity is primarily skeletal, due to an overgrowth of cartilage rather than to abnormal stresses on the skeleton by hypertrophied ligaments and diaphragmatic muscles. Observations which led us to this conclusion are presented.

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MANAGEMENT OF THE POSTRESECTION SPACE IN TUBERCULOSIS

I. Following Segmental and Wedge Resection

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Sunmount, N. Y.

THE past five years have seen a progressive increase in the popularity of simple excision for residual tuberculous lesions.[1] Their characteristic location in the apical, posterior segments of the upper and superior segment of the lower lobes makes resection of these segments a common procedure.[2-5]

Treatment of a restricted pneumothorax, occurring after segmental or wedge resections of the lung, is far from standardized. Few published reports dealing with this specific problem are available.[6] It would appear, however, that current methods of treatment are not uniformly satisfactory.

Since 1951, this institution, in cooperation with several others, has been engaged in the systematic resection of residual necrotic foci. Late in 1952, our attention was directed to the high relapse rate occurring in patients with annular shadows persisting after long-term chemotherapy had been discontinued.[7] Our surgical program then expanded to include not only the randomized closed-negative group, but the so-called open-negative and the usual number of open-positive and salvage cases.

As a result of this rather systematic and consecutive experience with resections, we have accumulated in the past three years a moderate number of cases on which the present study is based. This report covers a three-year period, Aug. 8, 1951, to Aug. 8, 1954, during which 308 thoracotomies were performed on 292 patients. This survey includes only segmental and wedge resections. Study of residual intrathoracic spaces of those patients who had lobectomies alone, lobectomies plus wedges and segments, or pneumonectomies will be presented in a future report.

Following resection of any volume of pulmonary tissue, no matter how contracted or functionless, a space is created which demands filling by adjacent parenchyma. In most instances of segmental and wedge resections, when the adjacent lung is relatively normal, space occupation is not a problem. The relative expansion of the remaining lung may or may not be associated with physiological deficit. Using current methods of estimating pulmonary function, Overholt and his associates[8-9] found very little over-all change compared to preoperative values. This is, in part, due to the fact that essentially functionless tissue is removed. Since, in the diseased area, ventilation is reduced almost equally with circulation and oxygen uptake, minimal adjustment is necessary in the postoperative period. J. J. Hirdes,[10] comparing the functional

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sequelae of lobectomy and segmental resections, found there was little difference. Factors, such as the occurrence of postoperative hemorrhage or the formation of a limiting fibrinous exudate over a raw lung surface, may be much more important than the actual amount of tissue removed.

Our definition of a postresection space is a local or restricted pneumothorax characterized by the absence of lung markings on x-ray. Since space occupation may be merely a mechanical problem, depending on the relative size of the residual space and ability of the remaining lung to expand, we have recognized two groups of space problems for the purpose of treatment and prognosis.

It has been stated that a persistent postresection space was tantamount to a bronchial fistula and was, therefore, always a positive space. Aspiration of many of these spaces has shown them to be essentially neutral, or even slightly negative, by manometric pressure determination. Failure of such space to fill during the early postoperative period suggests that either the adjacent lung is restricted by a plastic membrane or intrinsic fibrosis of the lung is preventing expansion. It has also been observed that segments adjacent to those involved with fibrotic tuberculosis may have undergone near-maximum distention and early emphysematous changes as a consequence of the contraction of their neighbors. If these segments are salvaged as space fillers, and we make every effort to retain disease-free tissue, their capacity for distention may be limited.

In addition to the absence of positive pressure in these spaces, clinical observations do not suggest bronchopleural fistulas. These patients are asymptomatic and a study of serial radiograms reveals that the majority of these spaces shrink and are eventually replaced by organized fibrinous exudate.

The second group of space problems are those with unequivocal evidence of bronchial fistula. As a rule, it is not necessary to utilize pressure determination in the recognition of these pneumothoraces. These patients have a chronic irritative cough, usually productive of streaking with frank old blood or sanguinopurulent sputum. The space, as seen by roentgenogram, rather than decreasing in size, characteristically balloons as much as intrathoracic relations will permit.

Approximately one patient out of five experiences transient space problems. We have had this complication in 61 patients (20 per cent) involving 308 pulmonary resections (Table I).

It is of interest to correlate the disease status of these patients prior to operation with the occurrence of a postoperative space. The majority had minimal to moderately advanced fibrocavitary tuberculosis. About half of the group had no evidence of remaining cavity by serial planigrams, while the remainder were operated on because of a persistent annular shadow. Again, the majority (49 or 83 per cent) had achieved sputum conversion prior to resection as a result of prolonged chemotherapy (6 to 9 months).

As most of this group had susceptible organisms, early or late tuberculous complications were not anticipated. Three patients (0.5 per cent) had positive cultures from a postoperative space. One followed a decortication in which the

TABLE I. INTRATHORACIC SPACE PROBLEMS FOLLOWING SEGMENTAL AND WEDGE RESECTIONS (308 OPERATIONS) AUG. 8, 1951 TO AUG. 8, 1954

NO. OF PATIENTS	CAVITY STATUS PREOPERATIVELY	SPUTUM BACT. PREOPERATIVELY	LOCATION OF SPACE		DATE OF SPACE APPEARANCE POSTOPERATIVELY	
61	Closed 29	Neg. 49	Apical pneumothorax	41	1st wk.	49
	Open 32	Pos. 12	Apical Hydropneumothorax	1	2nd wk.	9
			Apical Hemopneumothorax	8		
			Lateral pneumothorax	3	40 d.	1
			Basal pneumothorax	3	9½ mo.	1
			Total pneumothorax	2	3 yr.	1
			Combined (Apical and basal pneumothorax)	3		
			Recurrent combined	1		

patient had had a positive pleural effusion preoperatively. The remaining two were only single colony growths and did not present any clinical problem. Several of the most troublesome complications occurred in the closed-negative or so-called target point patients. It is our impression that the formation of a postresection space is largely a mechanical phenomenon and has no direct relation to the state of the patient's disease. We are in complete agreement, however, with the ever-present danger of bronchopleural complications in the open-positive patient with resistant organisms.[11]

Location of the persistent or recurrent pneumothorax was apical or apical posterior in 41 cases (67 per cent), paralleling the resected area (Table I). It may be mentioned that we routinely use two thoracotomy tubes. The tubes are placed on 12 to 18 cm. of negative pressure. The 8 cases of hemopneumothorax were of minor extent, consisting of apical hematomas or blood caps.

Resection of the superior segment or wedges from the lower lobe is prone to result in a lateral pneumothorax or basal supradiaphragmatic space. In 2 patients, the lung expanded well, but after seventy-two hours a total pneumothorax was noted. Both had fibrotic emphysematous lungs which were almost incapable of sealing peripheral leaks.

There were three instances of bilateral pneumothorax. Review of those cases in which bilateral resections were performed for all palpable disease suggests that the occurrence of a postresection space on the first side is not invariably accompanied by the same complication on the opposite side.

The typical apical pneumothorax (80 per cent) will be apparent during the first or second postoperaitve week. Almost daily x-rays facilitate early recognition and treatment of these spaces, particularly when, from the appearance and behavior of the lung at operation, they may be anticipated. Manifestation of a pneumothorax, attributed to the resection, is uncommon after the second postoperative week. Late development of a pneumothorax or hydropneumothorax is more likely to be associated with a bronchopleural fistula of tuberculous etiology. In our 3 cases of late fistulas, however, no evidence of tubercle bacilli was found in the drainage material from these spaces.

Fig. 1.

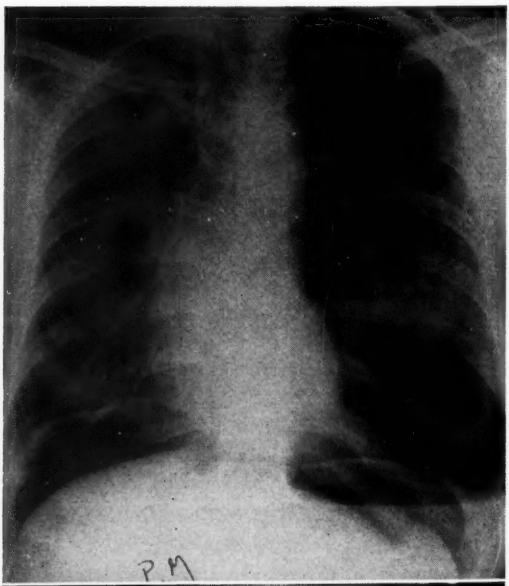

Fig. 2.

Fig. 1.—Neutral left apical space following segmental resection.
Fig. 2.—Same space three months later. No treatment.

Satisfactory management of the postresection space challenges the judgment of the surgeon and the various methods of treatment in current use.

As indicated, in the group showing no clinical evidence of a positive pneumothorax or bronchopleural fistula, this suspicion is confirmed by aspiration of the space. Furthermore, delay in space occupation may occasionally be anticipated from operative findings. In this situation, we feel that observation or careful neglect is adequate (Figs. 1, 2, and 3).

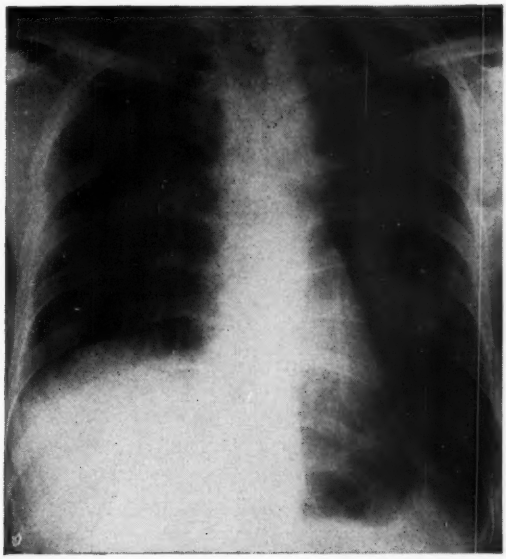

Fig. 3.—Left space almost obliterated. Small right apical postresection space.

On the other hand, the average space appears following removal of the original thoracotomy tube and represents a local tension pneumothorax, with a communicating fistula of varying size. This type of space, in our experience, is best treated with intercostal catheter, underwater-seal drainage on gentle negative pressure. Two-thirds of our series were successfully managed by single or multiple tube insertions (Table II).

Two adjunctive measures may be mentioned in connection with tube decompression of the local pneumothorax. The Trendlenburg position, if tolerated, is often utilized early in the postoperative period, with the hope that the remaining lung may adhere to the chest apex. A second measure, particularly useful for lateral-basal (supradiaphragmatic) or apical-basal spaces, is the induction of a pneumoperitoneum, provided the diaphragm is not fixed (Figs. 4 and 5).

We have been most hesitant to use enzymatic débridement in the treatment of a small hemopneumothorax.[12] Although it is claimed these enzymes do not cause fistulas, we are uncertain whether they contribute to their persistence.

Fig. 4.

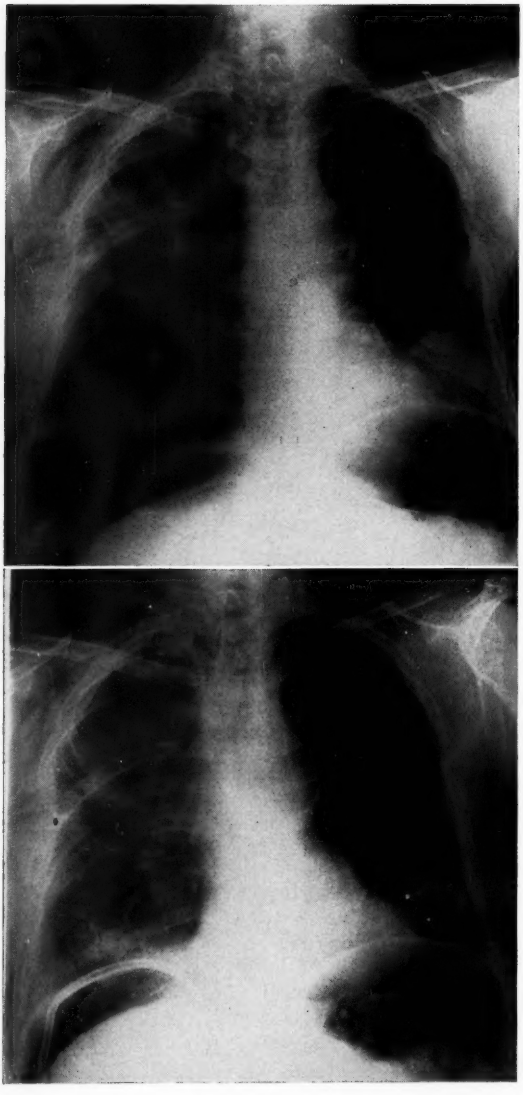

Fig. 5.

Fig. 4.—Right basal pneumothorax following superior segment resection.
Fig. 5.—Prompt resolution of space with intercostal tube aspiration.

Catheter drainage on underwater suction was successful in permitting spontaneous closure of the fistula and reduction of the space by the end of the week in 73 per cent of our group. At the end of this period, the presence of a fistula, virtually undiminished in size, raises several alternatives of treatment.

It is our impression that, after the second postoperative week, the boundaries of the local pneumothorax are relatively fixed. Subsequent organization of the exudate increases the rigidity of the space. At this time, many surgeons will elect to perform open thoracotomy, with attempted closure of the fistula, particularly if the leak is of appreciable magnitude. We have carried out secondary thoracotomy only four times. Three were satisfactory; the fourth recurred but responded to closed drainage. Although the fistula may be surgically closed, this does not guarantee obliteration of the space. In addition, an inlying drainage tube may invite pleural complications, both empyema and subscapular-space infections having resulted from attempted direct fistula closure.

TABLE II. TREATMENT OF SPACE PROBLEMS FOLLOWING SEGMENTAL AND WEDGE RESECTIONS

TYPE OF TREATMENT		DURATION		INFECTION (PREDOMINATE ORGANISM)		STATUS OF TUBERCLE BACILLI (N.T.A.)	
No treatment	6	1 wk.	35	Subtotal (space) empyema		Inactive	42
Thoracentesis	2	2 wk.	10	Proteus vulgaris	1	Active	4
Intercostal catheter	30	3 wk.	4	Friedländer's	1	Negative (In hosp.)	12
Multiple Intercostal tube	10	4 wk.	7	Mixed Proteus and tubercle bacilli	1	Deceased	1
Thoracentesis and tube	2	1½ mo.	2				
Secondary thoracotomy	4	2 mo.	4	Tubercle bacilli	3	No follow-up	2
Thoracotomy and Intercostal catheter	9	4 mo.	2	Hemolytic Staphylococcus aureus	11		
Pneumoperitoneum	6	5 mo.	3	Total (Staphylococcus) empyema	2		
Streptokinase-streptodormase	6						

Result of Space Therapy.—
Satisfactory, 50 Cured, 9 Unsatisfactory, 2

We have preferred to use the postresection thoracoplasty as an aid to space and fistula closure when intercostal tube decompression was unsatisfactory. Failure of intubation is usually apparent during the second to the fourth week.

There is definite morbidity associated with prolonged tube drainage. Intubation beyond two weeks almost invariably results in a low-grade infection of the space. Occasionally, pathogens such as *B. proteus* and *B. pyocyaneous* are present (Table II). These patients complain of intermittent fever and transient discomfort in the shoulder and anterior chest. Also, they are plagued with the well-known systemic handmaidens of a chronic local infection. The mental depression associated with attachment to a tube and bottle drainage for many weeks is difficult to estimate. As a rule, these patients complain of many apparently functional problems. Disregarding the elements of infection, prolonged intubation may accomplish gradual shrinkage of the space and fistula closure. Perhaps some of this is due to the layers of fibrinous exudate

resulting from the infection. Several spaces have reached the unsatisfactory point of diminishing returns when essentially no change is seen from week to week.

We have had the greatest success performing thoracoplasty after a two- or three-week trial of intubation when the space is still somewhat collapsible and is located in the apical posterior hemithorax. Thoracoplasty on top of a rigid space, walled with layers of connective tissue, accomplishes little, as with chronic, thick-walled parenchymal cavities.

The majority of space infections were nontuberculous and appeared to be a direct accompaniment of prolonged intubation (Table II). Hemolytic *Staphylococcus aureus* was the common organism, being in all cases penicillin resistant but usually sensitive to erythromycin. Since in most cases the space infections were adequately drained, the only concern was maintaining tubal patency. The infections with other secondary organisms or those in which tubercle bacilli were cultured usually responded to the time-tested principle of space obliteration and were not serious problems. Two total empyemas (Staphylococcus) were treated in the standard manner with drainage and subsequent partial thoracoplasty.

Results of space treatment have been classified as satisfactory, cured, or unsatisfactory (Table II). A small apical peripheral rim or cap pneumothorax which progressed to obliteration in several months was accepted as a satisfactory result. Of the unsatisfactory cases, both developed as a consequence of unduly prolonged intubation. Follow-up of this particular group was carried out to relate the occurrence of a space complication to possible late sequelae, and to the status of their tuberculosis (Table II). Four patients who are currently known to have active disease were reviewed, and no apparent relation to the pneumothorax was observed.

DISCUSSION

Proper management of the postresection space is necessary because of the ever present danger of infection and pulmonary hemorrhage. A chronic, undrained intrapleural space is similar in behavior to a parenchymal lung abscess with many of the same clinical symptoms. Even the neutral space, if unoccupied by lung, has infection potential. Since in most instances the presence of a pneumothorax is due to a bronchial fistula, obliteration of the space requires fistula closure.

The main contributing factor in the pathogenesis and persistence of postoperative bronchoalveolar leaks is the physical state of the remaining lung. Fibrotic lungs, with early emphysematous changes, are more likely to develop a postresection pneumothorax. Tube suction, in such instances, may achieve little toward space obliteration. Postoperative hematomas increase the anticipated incidence of bronchial fistulas, yet a most important factor is loss of the pneumatic function of the lung.

A postresection space, with a small fistula and residual lung capable of moderate expansion, will usually close within the first two postoperative weeks. Beyond this interval, although intubation alone may cause eventual fistula

closure, thoracoplasty or secondary thoracotomy may be a more reliable and earlier expedient. Secondary thoracotomy carries with it the danger of a disseminated intrapleural infection and associated subscapular space involvement. In addition, few thoracic surgeons will claim they have had any more than 50 per cent success in secondary bronchial stump closure. As with many surgical problems, there is no standard program, each requiring an individual solution.

SUMMARY

A review of 61 postresection pneumothoraces, occurring in 308 segmental and wedge resections, is presented. No relationship was found with the preoperative cavitary or bacteriological status of the patients. The location of the space paralleled the operative site of resected tissue. The majority of pneumothoraces appear within the first two weeks and respond to underwater suction if promptly diagnosed and intubated. A small number of spaces became chronic problems with pyogenic infection. In the presence of a relatively fixed space, early thoracoplasty or secondary thoracotomy may be required, in addition to tube drainage for ultimate definitive therapy. The occurrence of this complication does not appear to influence the future status of the patient's tuberculosis.

Addendum.—Since adopting the technique of approximating raw surfaces, we have had no postresection spaces in the past 75 thoracotomies.

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RECURRENT TRACHEO-ESOPHAGEAL FISTULA

(Case Report)

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THE literature of recent years has contained numerous reports of the successful surgical correction of congenital esophageal atresia with tracheo-esophageal fistula. A review of these reports indicates that recurrence of such fistulas is relatively rare, and in several of the larger series this particular complication does not appear. Several authors have suggested that localized suture line leaks might produce stricturing, and following this the re-formation of a fistula might be anticipated.

The few recurrent fistulas reported have been recognized in the early postoperative period and it seems that a rather striking clinical change in the patient has led to this recognition, usually in association with symptoms heralding a leaking site. Lyon and Johnson,[9] in 1948, reported the reopening of a fistula on the ninth postoperative day. Treatment by dilatation was unsuccessful. Autopsy revealed a congenital stricture distal to the site of the fistula which had been overlooked at surgery. Bigger,[3] in 1949, reporting on the treatment of 11 patients, mentions briefly one child who died following operation for a recurrent fistula which was recognized seven months after the initial operation. Potts'[10] report of 35 cases in 1950 does not mention recurrence of fistula. Recurrence has not been worthy of mention in the extensive experience of Gross,[4] though he does mention the fact that difficulties in swallowing and recurrent bouts of pneumonitis tend to plague this field of surgery. Abbott and Hopkins,[1] in 1951, reported 2 instances of recurrent fistulas in a series of 20 patients. One of these patients survived the second repair. Kopp and associates,[7] in a recent report from the University of Pennsylvania, reoperated for leaking anastomotic site in 6 patients of a series of 61, but recurrent fistula was not noted in any instance.

The rarity of recurrence of tracheo-esophageal fistula following primary repair of the congenital defects would seem to render the following case worthy of report.

CASE REPORT

History of Present Illness.—The patient was a 30-hour-old white boy, born at Walter Reed Army Hospital on May 27, 1952, at 7:38 A.M. Delivery was spontaneous. The mother was gravida iv, para iii. The serology was negative and, aside from polyhydramnios, her pregnancy was normal. The gestation period was 37½ weeks and the child weighed 6 pounds, 13 ounces. He breathed spontaneously. After three minutes in the oxygen hood, cyanosis and apnea were noted and tracheal aspiration and oxygen were necessary to revive the child. Rhonchi persisted throughout the day. The baby's temperature was variable throughout the day, rising to 101° F. At 6:00 A.M. he was offered glucose water.

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He took ½ ounce, promptly regurgitated, and turned deeply cyanotic. He seemed to have a great deal of difficulty in swallowing. An attempt was made to pass a Levin tube, but obstruction was met at approximately 3½ inches from the lips.

Physical Examination on Admission.—The patient was a well-developed, well-nourished, white, newborn boy with circumoral cyanosis. The hands and feet were slightly cyanotic. Otherwise the physical examination showed essentially normal findings.

Laboratory Data on Admission.—The red blood count was 4.8 million with hemoglobin of 15.1 Gm. On May 28, 1952, a fluoroscopic examination of the esophagus, by means of Lipiodol injection through a catheter in the hypopharynx, revealed an obstruction in the proximal esophagus at D_5. The distal end of the obstruction was conical in shape and apparently represented the end of the blind pouch. An upright film of the chest showed the lung fields to be clear (Fig. 1). The tracheobronchial tree was outlined by aspirated Lipiodol.

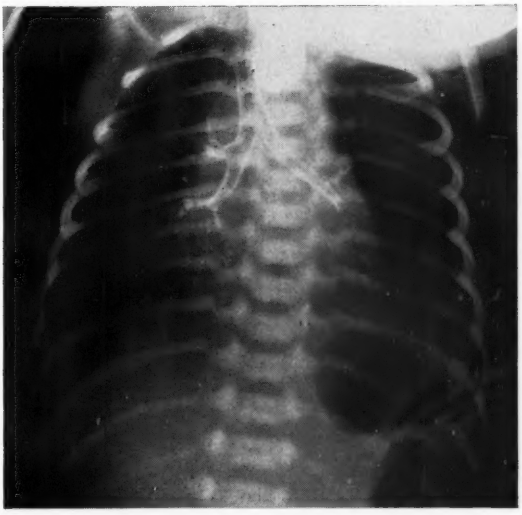

Fig. 1.—Initial x-ray, May 28, 1952.

The cardiac contour appeared to be within normal limits. The right lobe of the thymus was slightly enlarged and the remainder of the chest appeared to be within normal limits. There was a large gastric air bubble which indicated that the distal esophagus communicated with the trachea. There were no other significant findings. Impression: congenital atresia, proximal one-third of esophagus with tracheo-esophageal fistula involving the distal segment of the esophagus.

Operation.—May 28, 1952—Ether anesthesia was administered by pressure mask. A right thoracotomy was performed through a periscapular incision through the bed of the fourth rib. The extrapleural route was attempted, but the pleura was inadvertently opened in the apical area. After ligation and division of the azygos vein, the distal esophagus was found attached to the trachea just cephalad to the carina. The tracheo-esophageal junction was occluded with a Potts' clamp and the esophagus freed from the trachea. The tracheal opening was closed with a continuous suture of No. 5-0 arterial silk. The proximal blind pouch of the esophagus was then freed and pulled down into the right hemithorax. An opening was made in the most dependent portion of this pouch and end-to-end anastomosis between the proximal and distal esophagus was effected with interrupted sutures of No. 5-0 arterial silk. One small catheter was placed through the chest wall and attached to a water-seal bottle. The patient withstood the procedure well.

On May 31, 1952, a Stamm gastrostomy was performed under ether anesthesia.

X-rays.—June 2, 1952—A chest film revealed a triangular zone of opacity in the superior aspect of the right hemithorax, corresponding to an atelectatic right upper lobe.

Hospital Course.—The patient gradually gained weight but suffered repeated bouts of pneumonitis. The recurrent pneumonitis responded to antibiotics. There was no indication of a normal swallowing reflex and attempted oral feeding resulted in aspiration.

Six months after operation, in November, 1952, laryngoscopy revealed an infantile epiglottis without evidence of airway obstruction. False cords and true cords were normal and both sides were mobile. When the patient was in spasm the glottis closed tightly. This examination could not explain the aspiration of food. Impression: no disease of the larynx or hypopharynx. Repeat chest films made during this prolonged period of hospitalization continued to reveal atelectasis of the right upper lobe.

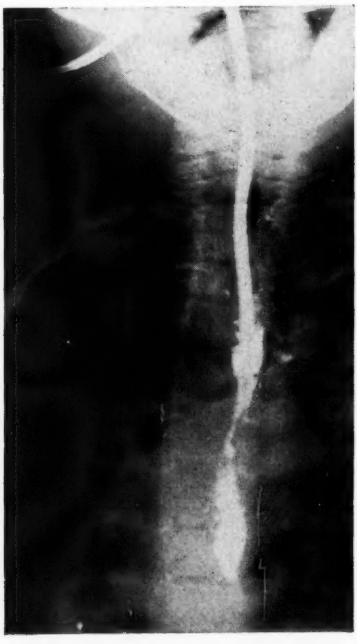

Fig. 2.—Lipiodol swallow, Dec. 11, 1952.

Lipiodol study of the esophagus performed on Dec. 11, 1952, revealed no definite evidence of fistula on fluoroscopic examination (Fig. 2). The cardiac shadow and mediastinal contents were retracted to the right. The films at this time showed small flecks of Lipiodol in the bronchus at the level of T_3. It was thought that these flecks were the result of aspiration, as communication between the trachea and esophagus was not visualized.

The patient continued to gain weight and appeared normal except for persistent inability to swallow.

Fluoroscopy was repeated on March 27, 1953. At this time Lipiodol entered the stomach without delay, but a small constant deformity, suggestive of traction diverticulum, was noted in the mid-esophagus just above the coryna.

X-rays taken on May 6, 1953, revealed atelectasis of the right upper lobe with the mediastinum drawn to the right. Otherwise the chest appeared within normal limits.

The child's general condition was good and he was discharged from the hospital to continue gastrostomy feedings. The repeated brief attacks of pneumonitis were attributed to the chronic atelectatic state of the upper lobe of the right lung, but the major item of interest throughout this prolonged period of observation was the child's inability to swallow. The interval follow-up of the patient reveals additional hospital admissions in 1953 for diarrhea and for dehydration. On each occasion the response to treatment was prompt. In November of 1953 the child was observed for 48 hours in the hospital because of blood-flecked vomitus. Outpatient follow-up was continued until March, 1954, when definitive study of the esophagus was deemed necessary to clarify the cause of the child's inability to swallow. Throughout the period from May, 1953, to March 31, 1954, the patient seemed to develop normally though slowly.

Fig. 3.—Demonstrates fistula, April 5, 1954.

The patient was hospitalized again on March 31, 1954. Physical examination at that time revealed a well-developed, well-nourished, small, alert, active boy weighing 18 pounds, 10 ounces with a temperature of 99° F. The tongue was maintained in a position of partial protrusion and the throat was normal. The chest was symmetrical with a healed periscapular scar on the right side. Auscultation of the chest revealed suppression of breath sounds over the right upper lobe, but no râles were heard. The heart was within normal limits by percussion and auscultation.

Laboratory Data.—The red blood count on admission was 3.6 million with a hemoglobin of 9.1 Gm. and the white blood count was 23,150 with 73 per cent neutrophils, 21 per cent lymphocytes, 2 per cent monocytes and 3 per cent eosinophils. Urinalysis was negative. On April 14, 1954, the white blood count was 32,600 with 10.5 Gm. of hemoglobin. X-ray taken on April 1, 1954, revealed considerable improvement in the appearance of the right upper lung field. Esophagoscopy was performed without anesthesia on April 2, 1954. A 5 mm. by 35 cm. esophagoscope was passed into the hypopharynx and

through a normal cricopharynx. At a distance of 15 cm. from the incisor teeth, the esophagus narrowed to a small opening postero-inferiorly and to the left, and to another opening visualized anterolaterally to the right. The opening postero-inferiorly was the larger and would admit a No. 16 French bougie which could be passed for approximately 5 cm., at which point resistance was met and further passage prevented. Normal mucous membrane appeared to cover both orifices. Diagnosis: atresia, congenital esophagus with possible residual broncho-esophageal fistula. X-ray studies were performed on April 5, 1954, and Lipiodol was introduced into the gastrostomy tube in the stomach (Fig. 3). The patient was placed in the Trendelenburg position. Manual palpation and multiple positioning were done, but no Lipiodol could be refluxed into the esophagus. A catheter was introduced nasally into the esophagus which passed readily down to the cardiac end and a small amount of Lipiodol was inserted. No abnormality of the lower end of the esophagus was noted. When the catheter was withdrawn to the level of the coryna, a small amount of Lipiodol was again injected. Almost immediately a small globule appeared in the right main-stem bronchus. Following a short delay the Lipiodol was seen in both main-stem bronchi. Impression: tracheo-esophageal fistula, probably just proximal to the coryna, with stricture of the esophagus at the operative site. The stricture was approximately 1 cm. long.

Operation.—April 15, 1954. The anesthesia was accomplished with gas-oxygen-ether administered via endotracheal tube. The patient's temperature was reduced to 93° F. by use of an ice mattress. A right thoracotomy was performed following excision of the existing periscapular scar and the fifth rib was excised. A generalized pleural symphysis was encountered. The right lung was mobilized by sharp dissection. Dissection of the mediastinum revealed a thick-walled fistulous communication arising from the posterior wall of the trachea approximately 1.5 cm. proximal to the carina and attached to the anterior wall of the esophagus at the site of the previous end-to-end anastomosis. The fistula was approximately 1 cm. in length. It was transected and the esophageal tracheal ends were closed with interrupted sutures of No. 3-0 black silk. A portion of the anterior mediastinal pleural flap and attached areolar tissue was placed over the tracheal suture line and anchored to the posterior tracheal wall, effecting interposition of tissue between esophageal and tracheal suture lines. The patient withstood the operation well. Rewarming was gradual over a two-hour period following the completion of the operation.

Postoperative Course.—Postoperative gastrostomy feedings were continued for ten days. Penicillin was given for fourteen days. The patient was started on purées on the tenth postoperative day and was gradually extended to a full diet which he tolerated well and without difficulty. He was gaining weight at the time of discharge on the eighteenth postoperative day at which time films revealed satisfactory clearing of the upper lobes of the right lung. Late follow-up films taken on Aug. 2, 1954, showed no evidence of tracheo-esophageal communication.

DISCUSSION

The recurrence of fistula following primary surgical treatment of esophageal atresia with tracheo-esophageal fistula is a rarity. Several factors might lead to the re-establishment of such a fistula: (1) a leaking esophageal anastomotic line with direct extension into the site of tracheal closure; (2) a direct contact between the adjacent anastomotic suture line of the esophagus and the suture line in the tracheal wall; (3) mechanical trauma resulting from early postoperative dilatation of the esophagus.

The contiguous position of the suture lines and a localized leak of the esophageal suture line would appear to have been the factors in the establishment of the fistula reported here. The patient's true pathology was masked by recurrent pneumonitis and a diagnosis of faulty deglutition for a period of

months until the existence of the fistula was proved. No doubt the repeated courses of antibiotics kept the child alive and also aided in tentatively substantiating the diagnosis of recurrent pneumonitis. The radiological studies performed during the first year of life were at best equivocal, but since a patent esophagus was demonstrated these tended to discount the possibility of a fistula. In view of the repeated prompt response of the upper respiratory symptoms to antibiotics, the equivocal x-ray studies, and the child's inability to swallow, it is understandable that such a clinical course might be interpreted, as in the case reported, in such a way as to confuse the need for esophageal surgery in infants.

Many accurate descriptions have been written dealing with the diagnosis of tracheo-esophageal fistula with esophageal atresia. The existence of a fistula without esophageal atresia or stenosis is not a familiar picture and consequently may be overlooked. Holt and co-workers,[6] in 1946 and Leigh and associates,[8] in 1951, directed attention to the difficulties which might delay or prevent the diagnosis of tracheo-esophageal fistula in the absence of esophageal obstruction. Such instances as have been reported exhibit similarity in the fact that they have masqueraded under the combined diagnosis of faulty deglutition mechanism and recurrent pneumonitis. Haight[5] has documented the details of a case in which fistula was suspected at birth, but the diagnosis was not confirmed until the patient was 4 years old, when surgical correction was carried out. Leigh and associates[8] in their report included 2 patients in whom the diagnosis of fistula without atresia was confirmed. Both these reports have suggested the use of additional radiological studies not generally used in the routine evaluation of tracheo-esophageal fistula with atresia. Holt[6] has been impressed with prone-position esophagograms while Leigh[8] was able to demonstrate fistulas with tracheograms made with the patient in a reclining position. Certainly, it seems justified to include these two radiological studies when evaluating a prolonged postoperative course of any infant following anastomotic surgery for tracheo-esophageal fistula with esophageal atresia. Such studies might be of great value in the diagnostic survey of any infant suffering repeated bouts of pneumonic infection, either with or without evidence suggestive of faulty deglutition.

The reports dealing with operative technique generally have not stressed any particular point designed to prevent the recurrence of fistula. Haight,[5] in 1948, recommended the use of a pedicle flap of mediastinal fat and areolar tissue to buttress the suture lines following the closure of tracheo-esophageal fistula. He called attention to the possibility of the recurrence of a fistula at the sites where the esophageal and tracheal suture lines were in close apposition. Albi and Gilchrist[2] in the following year, discussing tracheo-esophageal fistulas resulting from blunt trauma, suggested that the interposition of a mediastinal flap between the suture lines might prevent the recurrence of a fistula at the site of closure. The use of an interposed mediastinal flap should be a routine part of operative technique in the treatment of tracheo-esophageal fistula regardless of the etiology of the fistula.

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AN UNUSUAL CASE OF COARCTATION OF THE AORTA

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THE development in fetal life of the vascular trunk with the great vessels is based on a double anlage with subsequent reduction. The aorta caudal to the brachial arches becomes fused as an unpaired dorsal aorta, and the left fourth arch persists as the definitive aorta. For this reason congenital anomalies have a fairly high incidence. Eek,[1] Gross and Ware,[2] later Apley[3] have recorded these anomalies from a surgical point of view. Broadly speaking, they may be classified into 3 groups according to the presence of a double aorta, right aorta, or left aorta. Coarctation dominates in the last-mentioned group and may be observed alone or associated with other anomalies, the classic examples being bicuspid valve and cerebral aneurysm (Clagett and Jampolis[4]), as is also persistent ductus Botalli, which according to Abbott[5] is found in 10 per cent of the cases. This latter finding agrees with that made in materials from recent years (Knutson and associates[6]).

Coarctation mainly occurs in two groups, the adult type with a relatively short stenosis localized below the point of origin of the left subclavian artery, and below or at the level of the ligamentum arteriosum or ductus Botalli. The other main group, which has been termed the infantile or preductile type, is located above the ligamentum arteriosum, and is usually so long that surgical reconstruction is only possible with arterial graft (Gross and co-workers[7]). Frequently, the left common carotid artery and the left subclavian artery spring from the narrow site (Burford[8]). This offers poor or insufficient supply of blood to the left side of the brain, and the preductile type is not usually compatible with life for more than a few months.

We have recently had a case of coarctation of the aorta in which double stenosis was found, with a small intermediate chamber which was the point of origin of the left subclavian artery. Structural conditions were fairly well adapted for success in surgery without many serious difficulties. The immediate result was favorable, and we have therefore considered it justified to record the case here.

CASE RECORD

The patient was a 6-year-old boy, born at full term, who developed normally until he was 5 years old. Then he became unable to take part in play with other children, and he easily became sullen and low spirited ("ungovernable temper," Gerbode and Bourne[9]). He tired easily and his feet were cold. There was no cyanosis.

Clinical Examination.—His head was large. There was vigorous pulsation in the right radial artery, and also on the right side of the face. On the left side the pulse was feeble, but countable in the radial artery. There was an invisible, hardly perceptible pulse in the left

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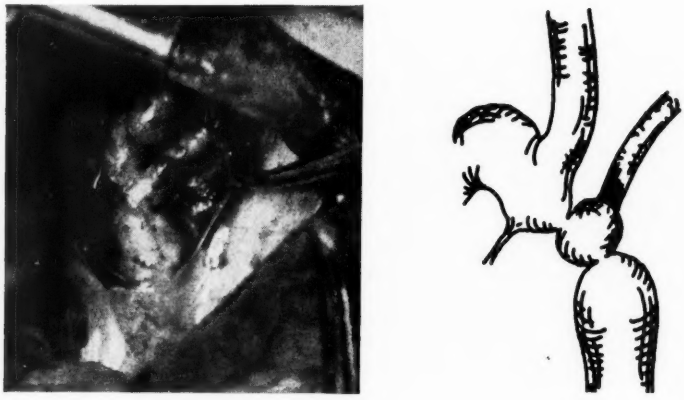
side of the neck and face; no perceptible pulse in the lower extremities, which were cold to the touch; no perceptible or visible collateral vessels on the chest. The blood pressure was 155/90 in the right arm, 100/70 in the left arm, and not measurable in the lower extremities.

A slight fremitus was felt over the heart, and a distinct systolic, blowing sound was heard, loudest in the second and third intercostal spaces at the sternal margin. The sound was heard upward on the right side of the neck, not on the left. There were no special findings by blood examination.

Electrocardiography.—Showed right axial deviation, hardly pathological.

Radiography.—Demonstrated a slightly enlarged heart, and increased width of the shadow of the vascular trunk with left outline as on a dilated subclavian artery. Strong pulsations in the ascending aorta and an impression in the esophagus at the level of the fifth and sixth ribs were considered to have been caused by a poststenotic dilatation of the descending aorta. Costal impressions were on the right side of the aorta—not on the left.

Because of the clinical and radiographic findings, it was assumed that the stenosis was located above or at the departure of the subclavian artery, and it was considered that the prospects of surgical correction were poor. As we would not deprive him of the chance, however, thoracotomy was performed on June 5, 1953.



A. *B.*

Fig. 1.—*A* and *B*, The anatomical findings after the exposure of the aortic arch and the great vessels.

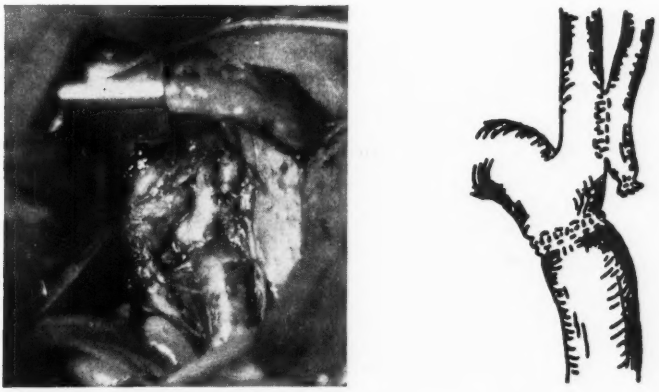
The chest wall held strikingly few collaterals. Two vessels arose from the top of the strongly pulsating aortic arch, one of almost the same caliber as the aorta proper (Fig. 1). Below the origin of this vessel, another vessel arose, which was of materially smaller caliber. Above and below the departure of this latter vessel, the aorta was greatly constricted, more so below the point of origin. The aorta proper was also narrower than usual at this point.

The pulsations in the largest vessel were very marked, those in the smaller vessel were very feeble. Both vessels had a parallel course, passing up toward the dome. No other vessels had their origin in the aortic arch, and it was thought that the greater vessel was the innominate artery with ample blood supply to the

right arm and the right carotid. The lesser vessel was assumed to be a trunk, common to the carotid and subclavian arteries, with correspondingly reduced pulsation in the peripheral regions of these vessels.

The duct had its origin above the upper constriction and was of medium dimensions. It was not felt to be patent by palpation. The development of the collateral circulation was strikingly weak with few and small intercostal arteries.

It was decided to perform a side-to-side anastomosis between the innominate and subclavian arteries in order to preserve some circulation while the anastomosis was placed, then to ligate the subclavian artery at the point of origin, and as the distance between the upper and lower constrictions was fairly short, to resect this part of the aorta, and make an end-to-end anastomosis in the usual manner after having first ligated the ductus Botalli. Due to the deficient collateral circulation, it was not considered justifiable to ligate the subclavian artery. As previously mentioned, the left carotid artery also originated from this trunk. The operation was performed as planned without essential difficulties (Fig. 2).



A. *B.*

Fig. 2.—*A* and *B*, The status after surgical reconstruction.

There followed immediately a marked improvement in the pulsation in the left arm and the left side of the neck and head. The day after the operation a distinct pulse was felt in both lower extremities, which were warmer than previously. The blood pressure in both legs was 140/90, and in both arms 120/80. The postoperative course was uneventful.

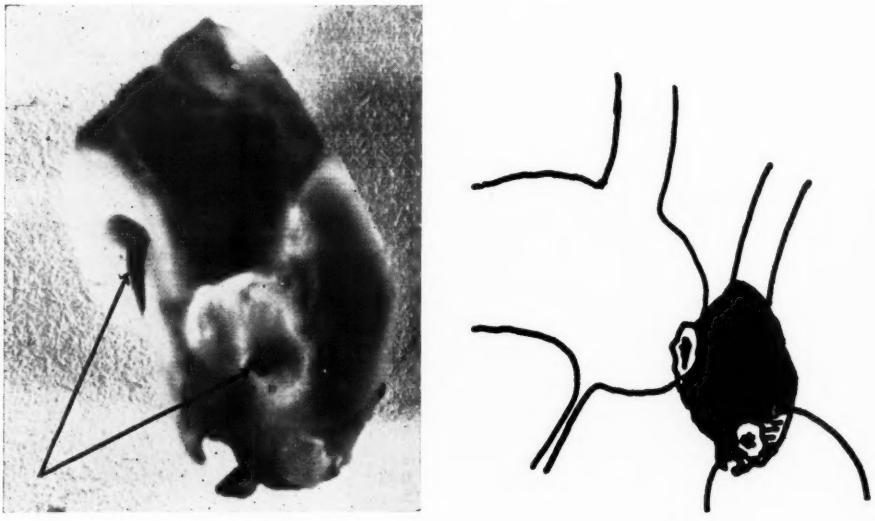
Control examination at the end of September, 1953, showed normal values of blood pressure in arm and leg, equal on both sides. Radiography showed reduction in the size of the heart.

DISCUSSION

Thus, in the present case there was the usual departure of the innominate artery. A vessel was present on the left side—a common trunk of

the common carotid and the left subclavian arteries. Above and below the point of departure there was stenosis of the aorta, most marked below (Fig. 3). The stenosis above, however, was so extensive that the blood pressure in the left arm was decidedly lower than in the right. Likewise there was no pulsation in the left side of the neck, while it was marked and vigorous in the right side.

Clarke and Dodrill[10] record a case with a common point of origin from the aortic arch of the four arteries (carotid and subclavian to both sides) with simultaneous coarctation. We have not, however, been able to find any case mentioned in the literature with a similar point of origin of a vessel as in the case just presented.



A. *B.*

Fig. 3.—*A* and *B*, The removed specimen of the aorta showing the stenoses.

Coarctation with unilateral hypertension and normal or low values contralaterally is well known (Weber and Knop,[11] Knutson and co-workers[6]).

In three cases of coarctation with lacking radial pulsation on the left side, Grishman and associates[12] succeeded in demonstrating by arteriography that there was atresia of the left subclavian artery concurrently. Gerbode and Bourne[9] gave an account of a similar case, in which the rudimentary subclavian artery was ligated, and the usual anastomosis made, after resection of the stenosed region. Holms and Love[13] recorded a case associated with stenosis of the right subclavian artery, in which the low values were found on the right side. We have, in our own series of coarctations, a similar case.

Ligation of the common trunk of the carotid and subclavian arteries without anastomosis might possibly have been undertaken, in our case without grave sequelae (Clagett[4]), though gangrene of the arm, after the Blalock operation, has been described (Lam,[14] Webb and Burford[15]). However, in due consideration

of the lack of collateral circulation, and of the fact that the carotid artery to the left side also took origin from this branch, we considered it advisable first to place an anastomosis between the innominate and subclavian arteries.

SUMMARY

A case of coarctation of the aorta is reported in which there was a double stenosis, above and below the departure of the common artery for the neck and left arm. Resection of the aorta was performed, combined with an anastomosis between the innominate artery and the common trunk of arteries to the left side.

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SURGICAL TREATMENT OF CORROSIVE STENOSIS OF THE THORACIC ESOPHAGUS BY SUPRA-AORTIC ESOPHAGOGASTRIC ANASTOMOSIS WITHOUT RESECTION

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A LARGE percentage of corrosive stenoses of the esophagus are in the province of the endoscopist and treatment consists of the following: (1) dilatation from above with esophagoscopic control, (2) dilatation along a string, and (3) retrograde dilatation with gastrostomy.

The disadvantages of all endoscopic procedures are the necessity for prolonged treatment, and the ever-present possibilities of mediastinitis from the trauma of dilatation, and of recurrent stenoses. Stenosis may occur and recur many years after the original injury.[1] Frequent dilatations and the chronic irritation from food passing through a corrosive stricture, even without obstruction, have a definite tendency toward the production of carcinoma. Benedict[2] reported from the literature a series of 236 cases of carcinoma of the esophagus of which 5.5 per cent followed benign strictures.

The failure of these methods, due to lack of cooperation of the patient, as well as extremely severe or old strictures, demands a surgical reconstructive procedure. Ante-thoracic reconstructions using skin tubes, small intestine, colon, or stomach are multiple stage procedures which require prolonged treatment and the results are uncertain and produce considerable disfigurement.

Intrathoracic reconstruction with resection of the esophagus has been successfully performed with the use of the colon, small intestine, and stomach through the left chest and more recently through the right chest. It occurred to me that leaving the esophagus in situ would cause little difficulty if adequate drainage for the segment were provided, and it was felt that if the segment were not exposed to chronic irritation the danger of carcinoma would be minimized.

Sweet[3] very aptly states: "Because of the extreme mediastinitis following lye burns of the esophagus, resection of the esophagus in these patients is unusually difficult and hazardous."

The following hazards are enumerated: (1) tearing of the tortuous esophageal arteries from the aorta, (2) bleeding from the azygous vein, (3) injury to the thoracic duct, (4) entering the opposite side of the chest, (5) production of vago-vagus reflex (this is probably due to a combination of anoxia and other facts but should be considered), (6) late vagus nerve sequelae, and the (7) prolonged operation time.

Moreover, by using a side-to-side technique, it was felt that the anastomosis would be more secure because the esophagus is not mobilized and that it would not tend to retract as with an end-to-end anastomosis. Also, the slitlike opening

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produced in a side-to-side anastomosis might tend to prevent regurgitation esophagitis and at the same time could be made sufficiently large to minimize the possibility of stenosis.

It was after consideration of these factors that this procedure was decided upon in the following case:

A 40-year-old white woman was first seen approximately 30 minutes after swallowing a cup of hot black coffee containing approximately 2 tablespoons of commercial lye. She was given vinegar and had profuse emesis. She was hospitalized with severe substernal pain, a white blood count of 20,000, and temperatures to 102° F. Parenteral fluids and antibiotics were used and after 10 days she was able to tolerate a clear liquid diet. She then disappeared until 5 weeks after the injury, when an esophagogram was taken and showed a severe stricture of the esophagus from the aortic arch to the stomach (Figs. 1 and 2). She was esophagoscoped and dilatation with soft rubber bougies was begun. She again cooperated poorly and was not seen until 4 months after the injury when a gastrostomy was performed and retrograde dilatations over a string were begun. She again left town and returned 4 months later (8 months after injury) with the gastrostomy closed and with a weight loss from 114 pounds to 73 pounds. She was again esophagoscoped and the stricture was found to be extremely firm, making surgical reconstruction imperative. She was prepared with blood, protein hydrolysates, and high caloric intravenous fluids. Before operation the protein was 7.2 mg., with albumin 3.62 mg., and globulin 3.50 mg.

Summary of Operative Procedure.—The patient was placed in right oblique position. The abdomen was opened, the incision incorporating the previous left gastrostomy incision. The stomach was mobilized by removing the spleen and transecting the gastrocolic ligament and left gastric artery near the celiac axis, care being taken to leave the vessels near the stomach intact as far as possible. The thoracic incision was then made from the angle of the fifth rib following the rib to the costochondral junction and extending inferiorly through the costal cartilages to join the abdominal incision. The lung was then retracted anteriorly and the esophagus very carefully freed above the aortic arch, care being taken to avoid injury to the right pleura or to the thoracic duct. A tube, placed in the esophagus through the mouth, expedited this procedure. The superior intercostal artery of the arch was transected to allow slight depression of the aortic arch. A Penrose drain was then placed around the esophagus in order to draw it into the field of operation. The lower end of the esophagus was clamped and transected and the normal esophageal opening into the stomach was inverted and sutured. The stomach was then brought up to the site for anastomosis which was easy to accomplish with slight mobilization of the left lobe of the liver. There was no question as to the blood supply of the stomach as the proximal end bled freely at the time of anastomosis. A posterior layer of sutures was then placed between the stomach and the upper esophagus and a one and one-half inch incision was made in both organs. The anastomosis was then completed in the usual manner with an outer row of interrupted cotton and an inner row of continuous intestinal catgut sutures. The tube in the esophagus was passed through the site of anastomosis into the stomach.

The distal transected esophagus was then anastomosed to the stomach by placing a small catheter 4 inches in length into the distal esophagus and inserting the esophagus into a stab wound in the stomach near the pylorus. A double row of sutures from the esophagus to the stomach were then placed. The stomach was then attached to the posteromedial thoracic wall with interrupted catgut sutures. The diaphragm was closed snugly about the stomach and the chest and abdominal wall closed in the usual manner. Underwater drainage was established through the ninth intercostal space. One million units of penicillin and 1 Gm. of streptomycin were instilled into the pleural and abdominal cavities before closure.

The patient had an unusually benign postoperative course. She was given Urecholine postoperatively and had no gastric retention, diarrhea, or other vagotomy symptoms, which were anticipated, although no pyloroplasty had been performed. Because of the extensive mobilization, the sympathetic fibers as well as the vagus were interrupted and this probably contributed to the lack of retention. The intrapleural tube was removed on the third postoperative day and the intragastric tube on the sixth postoperative day. She regurgitated the 4-inch

Fig. 1.

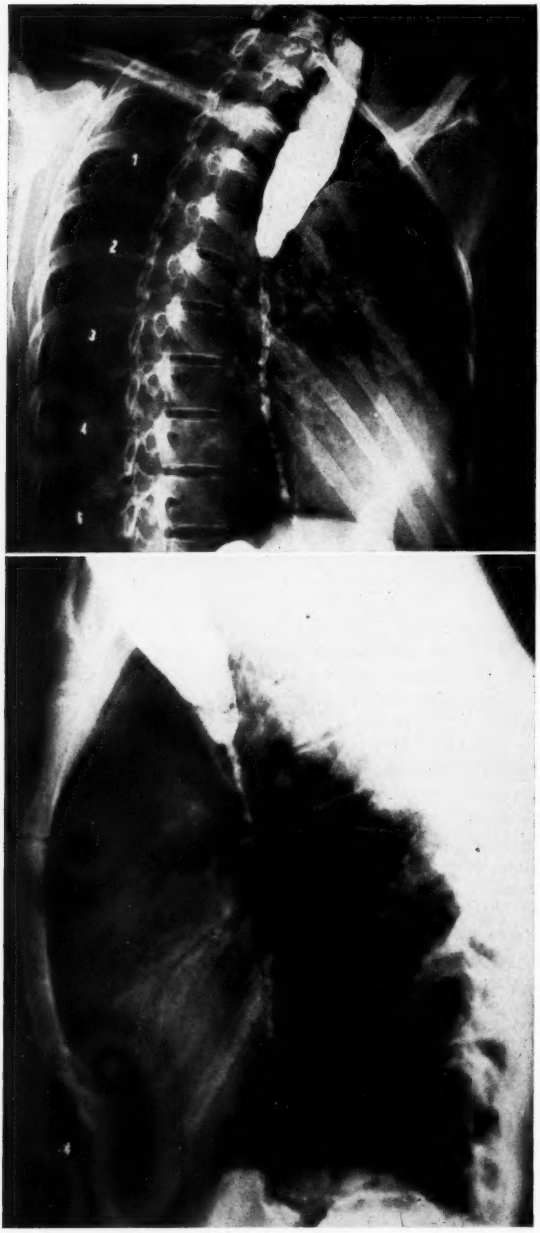


Fig. 2.

Figs. 1 and 2.—Oblique and lateral esophagograms showing stricture–numerals on oblique film were taped to skin for purposes of identification.

catheter from the lower esophagus several days after being discharged from the hospital. She was able to tolerate a soft diet on the seventh postoperative day and was discharged on the eleventh postoperative day. She rapidly gained weight and denied any regurgitation or other symptoms even when lying on her right side after a meal, with the exception of an occasional slight "soapy" taste (Fig. 3).

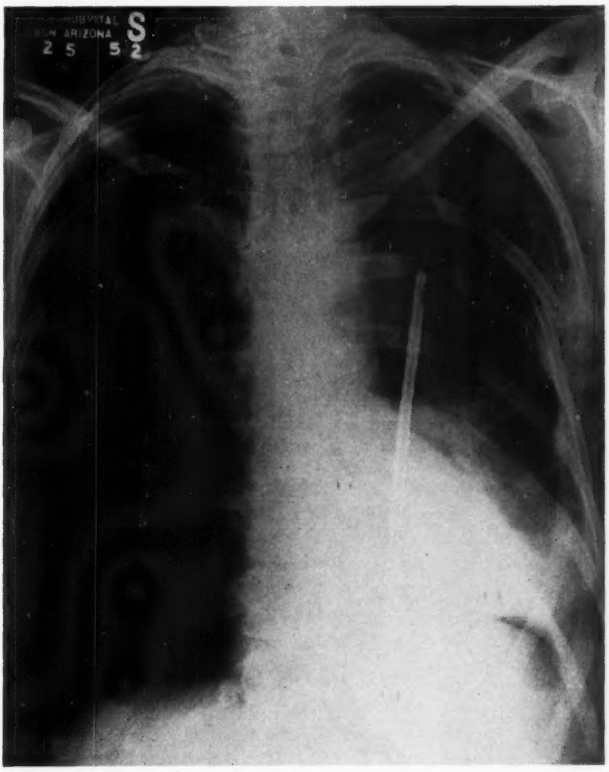
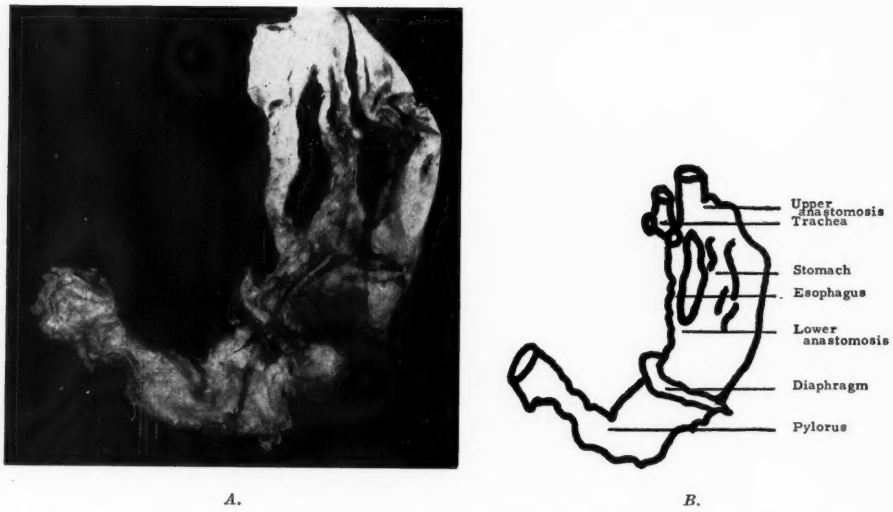


Fig. 3.—Chest x-ray taken on the fourth postoperative day. It shows the upper portion of the stomach at the level of the fifth rib, posteriorly which has been removed. The catheter in the stomach is the one allowed to remain in the lower esophagogastric anastomosis which has been extruded and is floating free in the stomach. It was later regurgitated through the mouth.

An interesting sidelight upon the case was that this patient was extremely enthusiastic after becoming gainfully employed, and regaining her normal weight, but approximately 14 months after her operation, she was attacked and killed by a gunshot wound in the head. I was able the examine the operative site at post mortem. The upper esophagogastric anastomosis was patent and adequate. The lung was normal with moderate pleural adhesions and adhesions to the stomach. The lower esophagus was completely obliterated so that a probe could not be passed from the stomach to the stenosed esophagus. There was no evidence of a collection of fluid or debris in the esophagus (Figs. 4 and 5).

DISCUSSION

Rapant and Hromada[4] reported 2 cases of esophagogastric anastomosis without resection in which the esophagus was mobilized to the aortic arch, thereby



A. *B.*

Fig. 4.—Photograph (*A*) of post-mortem specimen with explanatory diagram (*B*). Aorta has been removed.

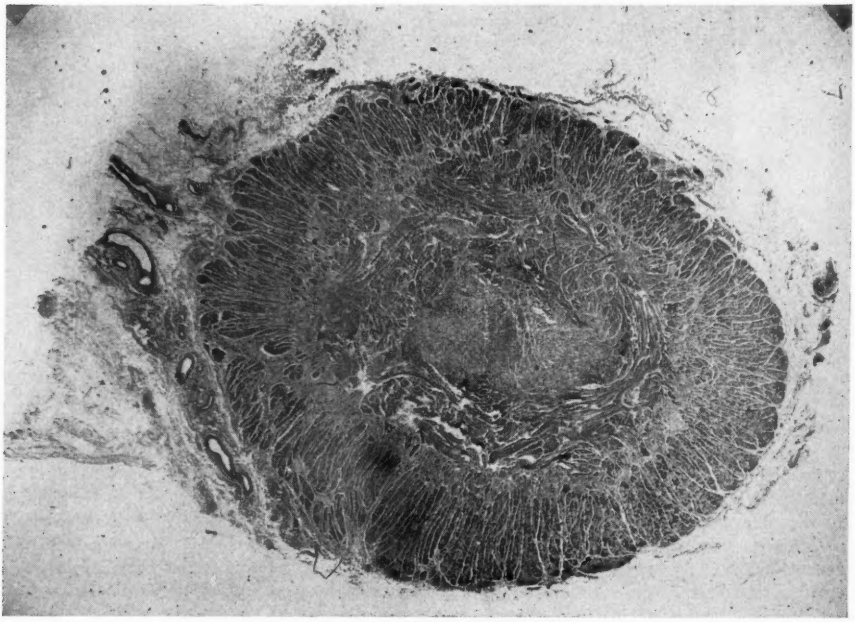


Fig. 5.—Photomicrograph of cross section of esophagus showing complete loss of epithelium and obliteration of lumen.

permitting upward traction on the stomach and its delivery behind the great vessels into the neck. This procedure requires considerable mobilization of the esophagus and does not appear to provide adequate drainage; it does have, however, the advantage of preserving the vagus fibers. They summarized the literature and are very enthusiastic about the results as compared with those obtained from extrathoracic or intrathoracic procedures with resection.

Ellis and Wilson[5] reported a case of lye stricture in a child, aged 3 years, with supra-aortic anastomosis after resection with a good result. The patient had a stormy postoperative course and developed atelectasis and later a pleural effusion.

Sweet[6] and Clagett and Taylor[7] also have reported cases of esophageal resection and high esophagogastric anastomosis. Even though the procedure described in the present article has been utilized in only one case I believe that the technique of leaving the esophagus in situ will result in less frequent complications and lowered morbidity as compared to anastomosis with resection.

SUMMARY AND CONCLUSIONS

A brief résumé of endoscopic and open surgical procedures available in the treatment of corrosive strictures of the esophagus is presented.

The advantages and disadvantages of leaving the esophagus in situ with intrathoracic esophagogastric anastomosis are given and the operative procedure summarized.

An unusual case is presented which was followed from the time of ingestion of lye through treatment by dilatation and successful surgery by this technique to complete recovery and later examination of the operative site at post mortem following death from an unrelated cause.

It is concluded that the supra-aortic esophagogastric anastomosis without esophagectomy as herein described offers a satisfactory solution to this difficult problem in selected cases.

I wish to thank Dr. D. J. Heim for assistance with the operation, Dr. Dennis Bernstein and Dr. Harry Neffson for endoscopy, Dr. H. D. Welsh for preparation of the x-ray photographs, Dr. L. Hirsch for preparation of specimen photographs, and Dr. M. R. Palmer for anesthesia.

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EXPERIMENTAL TRANSBRONCHIAL CARDIO-ANGIOGRAPHY

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INTRODUCTION

INTRAVENOUS angio-cardiography has been of little help in detecting left-to-right intracardiac shunts,[2] and it usually does not afford a detailed outline of the left side of the heart and the thoracic aorta. In an effort to improve the results of this method, procedures have been described which consist fundamentally either of injecting the contrast material under pressure into the pulmonary artery—for the visualization of the left atrium and ventricle,[4] or of taking numerous x-rays in a brief period of time during the injection of an opaque medium—for the diagnosis of interatrial septal defects.[5] However, in the opinion of its own authors, the former procedure gave no better results than those obtained with Robb and Steinberg's original technique; the latter seems to be a rather complex procedure requiring highly specialized radiological equipment.

It has already been reported[6] that clear pictures of the left ventricle can be obtained by introducing a contrast substance directly into that chamber. However, the fact that the left atrium cannot be outlined accurately places a definite limitation on this original method.

The purpose of the present study is primarily to extend the applicability of "cardio-angiography."[6] By injecting a contrast substance into the left atrium this chamber can also be properly visualized, together with the left ventricle and the aorta.

METHOD AND RESULTS

The procedure was carried out in a series of two normal dogs and four dogs with experimentally produced interventricular septal defects. These defects were created by inserting into the base of the interventricular septum a short, rigid plastic tube with a 4 mm. diameter bore.[7] The animals were studied from six to twenty-one months after the production of the defect, and physical findings as well as phonocardiographic and cardiac catheterization studies have confirmed the presence of a left-to-right shunt.[8]

After the animals had been anesthetized with pentobarbital sodium and placed in the right posterior oblique position, the left atrium was approached following a technique similar to that described by other investigators[1-3] for the measurement of left atrial pressures. Accordingly, a Chevalier Jackson bronchoscope was inserted into the trachea until the carina was visualized, and then a 15 gauge needle fitted to a saline-filled aspirating tube was passed through the

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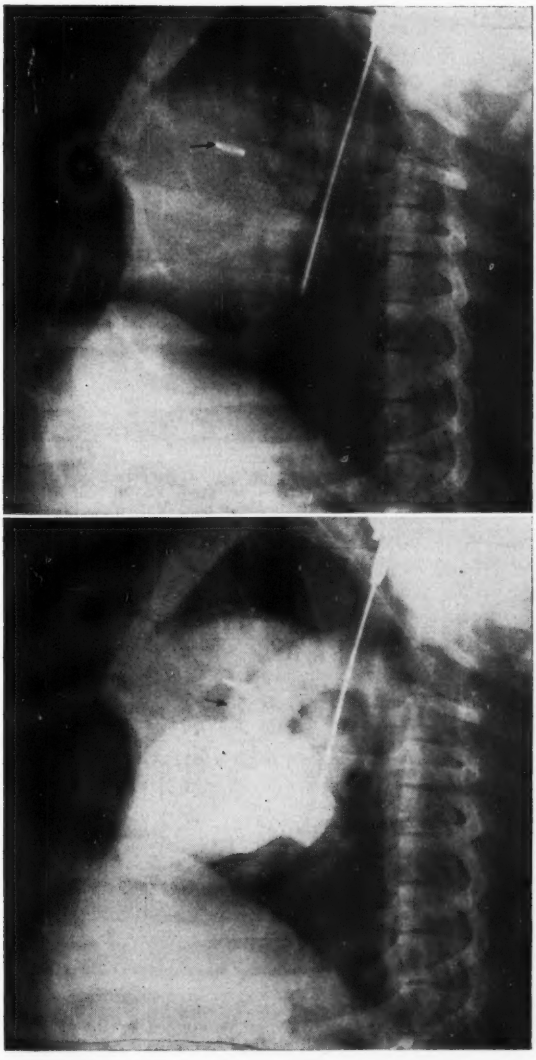
A.



B.

Fig. 1.—*A,* Chest roentgenogram of a dog with a subaortic interventricular septal defect produced with a plastic tube. The arrow points to the fine wire fixed to the proximal end of the device. The needle is seen in position, in the left atrium, ready for the injection of the contrast medium.

B, Chest roentgenogram of the same dog during cardio-angiography. The tip of the needle is seen in the left atrium. The arrow indicates the contrast material as it passes through the tube from the left to the right ventricle. The left atrium, the left ventricle, and the aortic arch are well outlined.

bronchoscope and inserted into the left atrium through the wall of the left main stem bronchus at a point 1 cm. from the carina. The location of the tip of the needle in the left atrium was confirmed by aspirating bright red blood with a syringe attached to the tube. Then, 70 per cent Diodrast equaling 1 c.c. per kilogram of body weight was injected rapidly by manual pressure. Films were taken simultaneously with the injection at a rate of four per second, using a Rigler-Watson rapid film changer.*

Electrocardiographic tracings revealed no significant changes either during or following injection of the contrast material.

Films of the normal dogs revealed good detail of the left atrium, left ventricle, arch of the aorta, and brachiocephalic arteries. In the dogs with interventricular septal defects the shunt of contrast material from the left to the right ventricle was detected in every case (Fig. 1, *A* and *B*).

COMMENT

This preliminary report is submitted because of the success of the procedure in a limited number of dogs, indicating its potential value in proper application to selected human subjects, in the elaboration of abnormalities of the cardiac septum, the left heart chambers, and the aorta. The diagnosis and localization of cardiac septal defects and the differentiation between mitral stenosis and mitral regurgitation may well be facilitated by this method. With the increasing progress of intracardiac surgery, the desirability of more exact methods for diagnosing these lesions has become apparent.

SUMMARY AND CONCLUSION

Cardio-angiography was performed on two normal dogs and four dogs with surgically created interventricular septal defects. The method used was based on the direct injection of contrast solution into the left atrium through the left bronchus under direct visualization through a bronchoscope. Good detail of the left side of the heart, the interventricular septal defects, and the aorta was obtained.

This method may have a valuable potential in the light of recent advances in intracardiac surgery.

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ANNUAL MEETING NOTES

The Thirty-fifth Annual Meeting of the American Association for Thoracic Surgery was held in Atlantic City, New Jersey, on April 24, 25, and 26, 1955. More than 750 physicians registered for the scientific sessions.

The following changes in the By-laws were voted at the Executive Session.

A section was added to Article IV of the By-laws as follows:

Section 8. Any member of the Association more than 60 years of age is excused from the attendance requirement of Article IV, Section 7, and upon his specific request may likewise be excused from the payment of dues.

Article V, Section 5, was amended so that the final sentence now reads as follows: "The Editor shall be elected from the Honorary, Active, or Senior members of the Association."

Article VI, Section 2, was amended to read as follows: The Membership Committee shall consist of seven (instead of six) Active or Senior members . . . and not more than five (instead of four) of the members may be reappointed to succeed themselves.

As a memorial to Dr. Howard Lilienthal, a Founder Member of the Association, a wooden gavel block was presented to the Association by his long-time pupil and associate, Dr. Arthur Touroff.

MEMBERSHIP NOTES

At a special meeting of the Council with the entire Membership Committee, the following clarification of the standards for election to Associate Membership was made. Copies of these standards which were accepted by the Association at its 1954 meeting were sent to each member of the Association last year.

A. Training:

1. Applicants in the United States must have a sound surgical training as evidenced by completion of the American Board of Surgery.
2. Applications in the United States must further have met the requirements of the Board of Thoracic Surgery. Canadian applicants must have Fellowship in the Royal College of Surgeons; be qualified in thoracic surgery or have passed the Royal College examinations for certification in thoracic surgery.

B. Professional Work:

1. High moral and ethical standards are required.
2. Applicant should be established in practice in his community or in a teaching position for *at least three years following training.* His professional work must be of the highest quality.
3. The applicant should have a major interest in thoracic surgery.
4. His professional conduct should be such that he has the support of the members of the Association living in the community.
5. The applicant should show evidence of future participation in meetings by making contributions to the medical literature, advancing the knowledge of thoracic disease, and by leadership in furthering thoracic surgery in his community.
6. Certification in thoracic surgery by the Board of Thoracic Surgery or by the Canadian Royal College of Surgeons *in itself does not mean fulfillment of the requirements* for election to Associate Membership. The other qualifications must be met.

C. Allied Specialists:

1. Members of the profession in allied specialties who are outstanding in their respective fields may be elected to membership in the Association according to the desirability for such specialists as determined by the Council.

D. Foreign Applicants:

1. Thoracic surgeons residing outside the confines of Canada and the United States and its territories are not eligible for election to the Association except as Honorary Members.

E. Exception:

1. Notwithstanding the above qualifications, in exceptional instances the Council may recommend for election a candidate who does not meet all of the above requirements.

The following changes in membership status were voted by the Association during its annual Executive Session:

In recognition of his outstanding contributions in the field of thoracic surgery and of his long continuous service to the Association, Dr. Evarts Ambrose Graham was elected to Honorary Membership.

The following Active members were elevated to Senior Status:

John B. Barnwell
George M. Curtis
Julian A. Moore
Harlan F. Newton
William F. Rienhoff, Jr.

The following Associate members were elected to Active membership:

Henry K. Beecher
David P. Boyd
George V. Brindley
Denton A. Cooley
Georges Deshaies
Howard A. Frank
Elliott Harrison
John F. Higginson
Alfred Hurwitz
John W. Kirklin
Roy G. Klepser
Rutledge Starr Lampson
Arthur M. Olsen
J. Karl Poppe
Charles B. Ripstein
Clifford F. Storey
Allan Stranahan
Wilson Weisel
George Willauer

The following candidates were elected to Associate membership:

Ralph D. Alley, Albany, N. Y.
Neil C. Andrews, Columbus, Ohio
H. William Clatworthy, Jr., Columbus, Ohio
George H. A. Clowes, Jr., Cleveland, Ohio
Oscar Creech, Jr., Houston, Texas
Albert C. Daniels, San Francisco, Calif.
Franklin H. Ellis, Jr., Rochester, Minn.
Joffre-Andre Gravel, Quebec City, Que.
Paul F. Hausmann, Milwaukee, Wis.
Nathan K. Jensen, Minneapolis, Minn.
Thomas N. P. Johns, Richmond, Va.
Victor H. Kaunitz, Buffalo, N. Y.
John L. Keeley, Chicago, Ill.
James E. Lewis, Jr., St. Louis, Mo.
William P. Longmire, Jr., Los Angeles, Calif.
Bertrand W. Meyer, Los Angeles, Calif.
Paul Nemir, Jr., Philadelphia, Pa.
Joseph L. Robinson, Los Angeles, Calif.
Gordon W. Thomas, St. Anthony, Newfoundland
Joseph L. Mangiardi, Freeport, N. Y.
Melvin M. Newman, St. Albans, N. Y.
Rodman E. Taber, Iowa City, Iowa

The following resigned from the Association:

Richard Davison

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